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LECTURES

ON

THE DISEASES OF

THE NERVOUS SYSTEM.

DELIVERED AT LA SALPÊTRIÈRE

BY

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TRANSLATOR'S PREFACE.

Although but recently published in Paris, the Lectures of Professor Charcot on Diseases of the Nervous System have already taken a place amongst the classic works of medical literature, and been translated into several Continental languages. When preparing for the following version, it was judged best to await the appearance of the second French edition; thus the reader, in exchange for some delay, has been enabled to obtain the work in its most correct form, enlarged by about one-sixth. It was found inconvenient to reproduce the ten plates appended to the French volume.

It is proper to mention that these lectures were reported and edited in French by Dr. Bourneville, editor of 'Le Progrès Médical,' whose name or initial will be found attached to several notes.

G. S.

Dublin.

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ON

DISEASES OF THE NERVOUS SYSTEM.

PART FIRST.

DISORDERS OF NUTRITION CONSEQUENT ON LESIONS OF THE BRAIN AND SPINAL CORD.

LECTURE I.

DISORDERS OF NUTRITION CONSEQUENT ON LESIONS OF THE NERVES.

SUMMARY.—Preliminary observations. Object of these lectures: they shall be devoted to those diseases of the nervous system, and of the spinal cord, especially, which are most usually met with in the Salpêtrière Hospital. Nutritive disorders consequent on lesions of the cerebro-spinal axis and of the nerves. These morbid alterations may affect the skin, the connective tissue, the muscles, the articulations, the viscera. Their importance in relation to diagnosis and prognosis. Nutritive derangements consequent on lesions of the peripheral nerves. Slight influence (in the normal state) of the nervous system upon nutritive action. Passive lesions of the nerves and spinal cord do not directly produce disorders of nutrition in the peripheral parts. Demonstrative experiments. Influence of the irritation and inflammation of nerves or of nervous centres on the production of nutritive disturbances. Nutritive disorders consequent on traumatic lesions of nerves, considered specially. They arise not from complete but from imperfect sections or from contusions, etc., of the nerve. Cutaneous eruptions: erythema, zona traumatica, pemphigus, "glossy skin." Muscular lesions, atrophy. Articular lesions. Lesions of the osseous system: periostitis, necrosis. Disorders of nutrition consequent on non-traumatic lesions of the nerves; their analogy with those which result from traumatic lesions. Nutritive disorders affecting the eyes in cases of compression of the trifacial by tumour. Inflammation of the spinal nerves, consequent on vertebral cancer, on spinal pachymeningitis, on asphyxia by charcoal fumes, etc. Cutaneous eruptions (zona pemphigus, etc.), muscular atrophy, and articular affections, which, in such cases, are developed in consequence of the neuritis. Anæsthetic lepra, leprous perineuritis, lepra mutilans.

GENTLEMEN: Never without emotion, yet never without great gratification, do I inaugurate, each session, the series of lectures which you have assembled to hear. On such occasions, indeed, I never fail to discover the friendly faces of former students, some of whom have attained professorial rank, and some of whom have already signalized their career by brilliant researches. Their presence affords me a great satisfaction, and I gladly seize the occasion to testify my gratitude.

It seems to me that the unusual number of those who have assembled here to-day is a convincing proof of the correctness of my belief when, five years ago, I ventured to maintain that this vast emporium of human suffering might one day become a seat of theoretical and clinical instruction, of uncontested utility.[1]

It is true, gentlemen, that the field of observation before us does not embrace the entire of pathology. But, taken for what it is, who shall complain of its extent, or say that it is not vast? On the one hand, it offers for our study the ailments of the aged, which call for a share of our attention. On the other hand, amongst chronic diseases, it exhibits, under conditions peculiarly favourable to research, and gathered together in numerous array, those diseases of the nervous and of the locomotor systems which are so common, and consequently so interesting to the physician—diseases the pathology of which has begun, within the last twenty years, to emerge from the deep darkness which had previously covered it.

As for myself, gentlemen, I have never doubted that the Hospital of La Salpêtrière was destined to become, both for the diseases of old age and for many chronic disorders, an incomparable centre of instruction. All that was required to realize this idea was that certain modifications should be made in the internal arrangement of this institution, and I am happy to inform you that circumstances, at present, seem wholly favourable to our views.

The authorities have already, without any solicitation, placed under our care wards containing nearly one hundred and fifty beds, where we may study all the forms of epilepsy and of the graver hysterical affections. The Director of the *Assistance Publique* has also formed the project of opening in this hospital a dispensary specially destined for patients suffering from chronic ailments, and a ward to which a certain number of them should be admitted, temporarily, to undergo treatment.

When all these elements of study shall have been classed and organized with a view to scientific investigation and clinical instruction, I have no hesitation in saying that we shall possess at Paris an institution which, of its kind, can scarcely have a rival.[2] I hope to have soon the happiness of seeing this plan realized in all its details. But if unforeseen circumstances should call me elsewhere, it would still yield me a deep gratification to see my successors crowning the edifice whose first foundations only I had been allowed to build.

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[1] This lecture was delivered in May, 1870.

[2] This project has, unfortunately, not yet been made a reality (September, 1874).

Gentlemen, your time is valuable, and I do not desire that this preamble should extend too far. It is time to come to the special subject of these lectures. I purpose, then, to devote this session to the study of those diseases of the nervous system, and especially of the spinal cord, which are most usually met with in this hospital. As I feel it would be objectionable to plunge at once into technical details, it seems to me suitable to invite your attention to a question of general interest, and one which we shall encounter at every step in the course of our studies.

I.

Lesions of the cerebro-spinal axis frequently react upon different portions of the body, and produce there, by means of the nerves, various disorders of nutrition. These secondary affections constitute one of the most interesting pathological groups, and I shall therefore devote several sittings to trace out for you the principal features of their history.

The consecutive lesions in question may affect most of the tissues and may occupy the most diverse regions of the body; thus, we may find them in the skin, the connective tissue, the articulations, the bones, and even the viscera. They generally present, at least at the beginning, the characteristics of inflammatory action. Frequently they play in the drama of disease but an accessory part, being simply added on to the usual symptoms, hyperæsthesia, anæsthesia, hyperkinesis, akinesis, motor inco-ordination, etc. But were it only for the interest they have, when considered from the standpoint of pathological physiology, they should not be neglected.

Occasionally, however, these lesions assume an unmistakable importance in the eyes of the clinical observer, either because of the serious ailments which they cause, or because of their value as regards diagnosis or prognosis. Allow me to offer some examples in support of this assertion.

Last year I pointed out to you, and I shall return to this symptom again, that the sacral eschar which is developed in the course of apoplexy from cerebral hemorrhage or from softening of the brain allows us to lay down a prognosis of almost absolute certainty. The sacral eschars, the affections of the kidneys and of the bladder, which are produced with such rapidity in certain acute diseases and in the exacerbations of some chronic diseases of the spinal cord, are often the immediate cause of death.

An arthropathy, arising in the course of locomotor ataxia, may deprive the patient of all future use of a limb which might otherwise have served him long.

Finally, these consecutive lesions of nutrition sometimes deceive the physician, who may mistake them for the disease itself. Such, for instance, are certain forms of progressive muscular atrophy which were formerly regarded as primary affections of the muscles themselves, and whose origin really lies in certain morbid alterations which have taken place in the gray matter of the spinal cord.

It would, I believe, be superfluous to multiply examples, for these observations should now suffice to indicate the interest which belongs to the study of such lesions of nutrition.

The power of producing, under certain morbid conditions, lesions of nutrition, in the peripheral parts of the body or in the viscera, is not an attribute of the brain and spinal cord alone. These centres share the privilege with the nerves which radiate from them. And it is to be observed that the consecutive affections produced by protopathic lesions developed in the most widely different regions of the nervous system present most remarkable analogies, in spite of some specific differences. Hence when the physician's attention has been called to such affections it is often a question of extreme difficulty to determine what portion of the nervous system was originally affected, and what is the true cause of the trophical lesion.

This consideration has induced me to limit our study to the lesions which are assignable to cerebral or spinal causes alone. These shall be, if you will, our objective point; but it seems useful to draw out, in parallel lines, the history of those trophical troubles which appear in consequence of lesions of the peripheral nerves. Is it not, indeed, one of the greatest advantages of the comparative method that it creates light by contrasts? In order to bound our field of study, we shall, however, only take into consideration those nutritive disorders which appear in the *peripheral domain of the suffering nerve.* The trophical changes which take place in consequence of reflex action, in a region more or less remote, and within the domain of nerves which have undergone no primitive lesions, constitute undoubtedly an interesting subject, but one which deserves to be treated specially.

II.

In hearing me speak, gentlemen, of the nutritive disorders which arise under the influence of lesions of the nervous centres or of the nerves, most of you, I am sure, have been immediately reminded of the corresponding problem which is debated in normal physiology.

There is nothing better established in pathology (as I hope to demonstrate) *than the existence of trophical troubles consequent on lesions of the nervous centres or of the nerves.* Nevertheless, you are aware that the most advanced physiology teaches that, in the normal state, the nutrition of different parts of the body does not essentially depend upon the influence of the nervous system.

These statements appear contradictory, but the opposition is only in appearance and not in reality. This I shall endeavour to prove, and with that object I have to ask your permission to make a short incursion into the domain of experimental physiology.

You are aware that in order to show that the chemical acts of molecular renovation, which constitute nutrition, are not immediately dependent on the action of the nervous system, many kinds of arguments are adduced:—

1. The most complex acts of nutritive life take place in certain organisms without the intervention of a nervous system. Plants, for instance, and some of the lowest animals, such as certain protozoa, though unprovided with nervous systems, manifest great vital activity. Does not the embryo, it is also asked, perform all the acts of organic life, at a period when it as yet possesses no nervous element whatever?

2. They base another argument on the fact that certain tissues, even in the superior animals, are totally devoid of nerves and vessels. As instances, they refer to the epithelial layer and to cartilage, which if placed under pathological conditions will become seats of cell-proliferation—a plain proof that nutrition can take place there in a very energetic manner.[1]

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[1] . . . "The whole organic life of animals, *i. e.*, everything which goes on in them without the intervention of any sensation, or other mental act, may go on without the intervention of the nervous system and stands in no relation of dependence to any change in nervous matter; just as the corresponding functions of circulation, nutrition, secretion, absorption, go on in equal perfection in the lowest class of animals where no nerves are detected, and in the whole vegetable kingdom, where there is no plausible reason for supposing that nerves exist; . . . the nervous system lives and grows within an animal as a parasitic plant does in a vegetable."—'British and Foreign Med.-Chir. Review,' vol. iii, 1837, pp. 9, 10; and Carpenter, 'Principles of Human Physiology,' Philadelphia, 1855, p. 59.

The following is a succinct analysis of an essay in which M. Charles Robin has quite recently expounded the prevalent ideas of the present day in reference to the far from prominent *rôle* which the nervous system plays in the work of nutrition: "Those chemical acts which, in a living organism, constitute molecular renovation, otherwise called nutrition, are not under the direct influence of the nerves. There can be no question here of an influence of nerves over tissues, comparable to that of electricity upon chemical action. There exist no nerves which extend over the extra-vascular anatomical elements, such as the epithelium, like those nerve-tubes which proceed and are applied to the muscular fibrils. The cause of the movement of nutrition lies in the anatomical elements themselves. In plants, where no nervous system is found, we see the tissues suddenly swell, the cells increase and multiply. In the embryo, cells are formed, and increase and multiply before the appearance of any peripheral nerve-element. Nutrition is, therefore, a general property of anatomical elements, be they animal or vegetable. Secretion itself is a property inherent in anatomical elements, as

3. Finally, arguments bearing more directly on the subject are drawn from the arsenal of experimental physiology. You know that, after section of the nerves supplying them, and even when the spinal cord has been destroyed, the peripheral parts of the body, such as the muscles or the bones of a limb, will continue to live and be nourished for a considerable time, almost as efficiently as though they were under normal conditions. In such cases, lesions of nutrition do not make their appearance until a comparatively long period has elapsed. Even then they are almost always purely *passive*, and seem, in reality, due to the state of inaction to which the parts are condemned in consequence of the suppression of nervous influence. This belief is supported by the fact that lesions, displaying similar characteristics, present themselves when limbs are kept in a state of immobility, though the nervous system be not directly implicated. Such passive lesions, which we shall meet with in different paralytic affections, have nothing in common with the special trophical lesions which engage our attention. Generally they can be distinguished from them, objectively, by certain particular signs.

The special lesions are almost always characterized, at some period of their evolution, at least, by evidence of phlegmasic irritation. From the commencement they usually take on the appearance of inflammation; and they may, as we shall see, issue in ulceration, gangrene, and necrosis. There is, besides, one characteristic common to most of them, and that is the great rapidity of their development, after the lesion of nerves or nervous centres which provokes their manifestation. Sometimes they make their appearance with incredible quickness. Thus we frequently see eschars visible on the sacrum, the second or third day after the accident, in certain cases of fracture of the backbone, with compression and irritation of the spinal cord.

It may therefore be laid down as a general rule that there is a striking distinction between *passive lesions*, resulting from functional inaction alone, and *trophic disorders* which follow on certain lesions of the nervous centres. The former are slow of production, and usually manifest no symptom of inflammation; the latter often suddenly break out and generally present, at least at the commencement, more or less notable signs of phlegmasic irritation.

De Blainville and A. Comte have observed. In the lower animals, and in the case of animal grafts, it is evident that the nutrition of tissues is independent of the nervous system." "Disorders of secretion and of absorption, induration, softenings, hypertrophies, and other alterations consecutive on nerve-lesions, are a consequence of perturbations of the circulation through the medium of the preceding (vaso-motor) nerves, directly affected by reflex action, and are not a consequence of the action of nerves which should, like electricity, have an influence over the molecular or chemical acts of assimilation and dis-assimilation in a zone of a certain extent beyond their surface."—'Journal de l'Anatomie,' etc., 1867, pp. 276–300.

Allow me, gentlemen, to remind you, briefly, of some of the experiments to which I have just alluded, and which tend to demonstrate that the nerves and spinal cord have no direct immediate influence upon the nutrition of peripheral parts.

1. One of the first of these relates to the section of the ischiatic nerve in mammalia. Schroeder van der Kölk, who was one of the earliest to make the experiment, attributed the disorders of nutrition which followed, with some rapidity in such cases, in the corresponding member, to the abolition of the action of the nervous system in consequence of the section. M. Brown-Séquard repeated the experiment, in 1849, on guinea-pigs and rabbits, and succeeded in showing that the trophical troubles which follow in the course of a few days, and which consist of tumefaction of the extremity of the member, ulceration of the toes, loss of the nails, only make their appearance because the animal is no longer able to preserve the limb, now devoid of movement and sensation in consequence of the section of the ischiatic, from the action of external influences, such as contact with the hard rough ground over which it is dragged. When the animal experimented on was placed under proper conditions, confined in a box, for instance, the bottom of which was covered with a thick layer of bran, there was no modification of nutrition to be remarked in the paralyzed member, except a more or less perceptible atrophy which, however, only made its appearance slowly in the course of time.[1]

This atrophy, which follows the section of the ischiatic nerve, evidently results from the functional inaction to which the paralyzed limb is condemned. It affects not only the muscles, but also the bones and the skin, as J. Reid has already remarked. It will not be produced, even when the section has been complete, if, following the example of the physiologist quoted, you take care to pass a galvanic current daily through the muscles of the paralyzed member.

2. The complete section of the trifacial nerve, made within the cranium, presents results perfectly in keeping with those produced by section of the ischiatic. You are aware that the lesions of the eye which are found in animals subjected to this operation, and which were formerly considered by some physiologists as the consequence of the abolition of the nutritive influence of the trifacial, have, since the experiments of Snellen in 1857 and those of Büttner in 1862, been recognized as resulting from the consecutive anæs-

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[1] Brown-Séquard, "Sur les altérations pathologiques qui suivent la section du nerf sciatique," 'Comtes-rendus des Séances de la Société de Biologie,' t. i, 1849; and 'Experimental Researches applied to Physiology and Pathology,' New York, 1863, p. 6.

After the section of a mixed nerve the atrophy of the muscles does not generally begin to show itself, in man and mammalia, by a slight emaciation, until the end of about a month. At the close of the second month it is more marked; it is very evident at the end of three months.—Magnin, *Thèse de Paris*, 1866, p. 19.

thesia which exposes the parts deprived of sensation to all kinds of traumatic causes. If the eye be protected after the section of the nerve, either by Snellen's method of tying the still sensitive ear of the same side in front of it, or by Büttner's plan of covering it with a piece of thick leather, the trophical troubles will not make their appearance in the cornea. A certain amount of neuro-paralytic hyperæmia in the iris and conjunctiva is, in short, the only phenomenon observable, after section of the trifacial, when the eye has been properly protected.[1]

3. With respect to the spinal cord it seems demonstrated that a complete transverse section, or even its destruction for a certain length, when resulting in no considerable inflammation of the organ, is not immediately followed by troubles of nutrition in the paralyzed members. M. Brown-Séquard has shown that the ulcerations which appear, rather quickly, in the vicinity of the genital organs of mammalia and birds, after complete transverse section of the cord, are not direct consequences of the absence of nervous influx. They are produced by the prolonged pressure, and the contact of fecal matters and decomposed urine, to which these parts are exposed.

The hinder limbs of a young cat, which survived for nearly three months the complete destruction of the lumbar region of the spinal cord, were seen to develop in a normal manner. The functions of organic life seemed to proceed there in due accordance with physiological order; the secretion of claws and hair went on as in a healthy and uninjured animal.[2]

According to Valentin, when the posterior portion of the spinal cord has been destroyed, in mammalia and frogs, you will find the electrical contractility of the muscles of the hind members persist until death supervenes, that is to say, for several weeks or even several months after the operation.[3]

To sum up: in those animals whose spinal cords have been completely divided transversely, or extirpated in part, ulcerations and even eschars may form, principally in those regions subject to pressure; but it is always possible to attribute these lesions to the anæsthesia and paralysis, in consequence of which the animal lies constantly in contact with its excrements or unwittingly wounds itself, when dragging about its paralyzed members. As to the atrophy which supervenes, in the long run, in the paralyzed limbs after this operation, it arises solely, as in the case of the section of the ischiatic nerve, from the functional inertia to which they are condemned.

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[1] See the experiments of M. Schiff relating to this subject in the thesis of M. Hauser, entitled 'Nouvelles recherches relatives à l'influence du système nerveux sur la nutrition,' Paris, 1858.

[2] Brown-Séquard, *loc. cit.*, pp. 14, 15, 16.

[3] Valentin, 'Versuch einer Physiologischen Pathologie der Nerven,' 2 Abth., p. 43, Leipzig, 1864.

It follows from all these facts, which experimental physiology offers, that the abolition of the action of the nervous system, whether determined by complete section of the peripheral nerves or by destruction of a portion of the spinal cord, produces no other nutritive disturbance in the anatomical elements of the paralyzed members than what would be caused in the same elements by the influence of functional inertia, or prolonged inaction alone.

The discovery of the vaso-motor nerves and of the effects which follow the paralysis of these nerves was not destined to modify this formula, in any essential manner. It is in fact demonstrated at present, that neuro-paralytic hyperæmia, however far it may go, is never of itself alone sufficient to cause an alteration in the nutrition of tissues. Undoubtedly, this hyperæmia, as M. Schiff has pointed out, creates a certain predisposition to inflammatory action, which may supervene either spontaneously (to all appearance, at least) in the diseased animal or in consequence of irritative causes which would be comparatively trifling in a healthy organism. But lesions of nutrition of neuro-paralytic origin are nowise comparable to the trophical troubles which form the special object of our study, —they constitute a class apart. The latter, as we shall frequently have occasion to observe, may develop and accomplish their evolution without being preceded or accompanied by any of the phenomena which betray the paralytic state, or the contrary condition, of the vaso-motor nerves. At present we shall dwell no longer upon this subject, which we shall have an opportunity of referring to hereafter.

III.

If lesions, whose consequence is the abolition or suspension of the action of the nervous system, are impotent to produce in distant parts other nutritive disturbances than those attributable to prolonged inaction, *it is not thus as regards lesions which determine either in the nerves or nervous centres an exaltation of their properties, an irritation, or an inflammation.*

That, gentlemen, is a proposition of capital importance: it controls, in fact, the question which engages our attention. Although long since discovered by M. Brown-Séquard the principle upon which it reposes is still, if I do not mistake, too frequently overlooked both by physiologists and by pathologists.[1] We shall find, in due time and place, that human pathology presents many facts and decisive arguments in support of this proposition. On the other hand, we shall have less frequently to quote the results of experiments on animals. The especial reason of this paucity lies, undoubtedly, in the fact that the nervous tissue of animals seems much better able than that of man to resist the influence of the diverse causes of irritation and inflammation. All experimenters are aware that even the most serious traumatic lesions of the spinal cord or of the peripheral nerves do not readily produce, in the case of most animals, a myelitis or a neuritis, having some duration, which could be considered comparable with those developed so quickly in man, after the very slightest lesions.

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[1] "Note sur quelques cas d'affection de la peau, dépendent d'une influence du système nerveux," par J. M. Charcot, suivies de "Remarques sur la mode d'influence du système nerveux sur la nutrition," par le docteur Brown-Séquard, 'Journal de Physiologie,' t. ii, No. 5, p. 108, 1859.

The experiments which go to show that irritative lesions of the nerve-tissues are capable of determining various trophical troubles in the parts they supply, are, as we have said, few in number. They relate almost exclusively to the fifth pair.

The following is an abridged account of an experiment of Samuel:

In the case of a rabbit, two needles are applied to the Casserian ganglion and an inductive current produced; immediately ensue a more or less marked contraction of the pupil, and at the same time a slight injection of the vessels of the conjunctiva. The lachrymal secretion is greatly increased. The sensibility of the eyelids, conjunctiva, and cornea is augmented. After the operation, the contraction of the pupil persists, though not to the same extent, and the hyperæsthesia is still further increased. Inflammatory action generally sets in at the end of twenty-four hours; it increases in intensity during the second and third days and then gradually diminishes. All the stages of ophthalmia may be observed, from the slightest conjunctivitis to the most intense blennorrhœa. The exaltation of the sensibility still proceeds, and the hyperæsthesia may rise to such a degree that, at the slightest touch of the eye, the animal is seized with general convulsions. On the cornea a general opacity develops, and we find, besides, sometimes little exulcerations, sometimes a solitary oval-shaped ulcer occupying the middle portion of this membrane. In one case a small purulent collection formed in the anterior chamber. Hyperæmia excepted, no pathological alteration of the iris, neither adhesions nor changes of colour, are ever observed.

In every instance, hyperæsthesia of the ophthalmic branches of the fifth pair is specifically remarked. Hence it is plain that we cannot here, as in the experiments of Snellen and Büttner, invoke the aid of anæsthesia in order to explain the trophical troubles supervening in an imperfectly protected eye.[1]

After an unsuccessful attempt to divide the trifacial in a rabbit, Meissner observed certain remarkable lesions of nutrition to ensue in the eye, which had preserved its sensibility. The author carefully points out that *these lesions were produced without having been preceded by any sign of neuro-paralytic hyperæmia.* A post-mortem examination revealed that the (internal) median part of the trifacial alone had been wounded by the instrument (a neurotome).[1] Schiff also cites four cases, in support of Meissner's observation, of partial lesions of the trifacial in the cranium, which were followed by inflammation of the eye, although its sensibility persisted.[2]

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[1] Samuel, 'Die Trophischen Nerven,' Leipzig, 1860, p. 61.

In Samuel's experiment trophical troubles arose in the eye, in consequence of faradaic irritation of the fifth pair. May we not infer that, in the experiments of Meissner and Schiff, the lesions of the eye were caused by phlegmasic irritation developed in the nerve in consequence of the imperfect section? In support of this opinion, I would remind you that incomplete sections, in man, are much more likely than complete sections to give rise to irritative action. This fact has long been familiar to surgeons. We may suppose that it holds good, at least to some extent, in the case of animals as well as of man.[3]

Let me place, side by side, with these facts several observations recorded in reference to the human organism, to which I shall afterwards recur. They relate also to the trifacial nerve. Like the preceding experiments, they show that irritative lesions of this nerve, spontaneously developed, may also, without being followed by anæsthesia, give rise to very striking nutritive disorders in the eye.

A woman, aged 57, whose case has been noted by Bock,[4] experienced, for about a year, violent pains in the right side of the face. Though intermittent at first, they became afterwards almost continuous. The sensibility of the face never completely disappeared; slight pressure was, indeed, scarcely felt, but if the pressure was increased, it brought on acute pains. The conjunctiva of the right eye was injected. The cornea, slightly opaque all over, presented a hypertrophic ulceration in its lower part, of about two lines in length. Afterwards, the ulceration increased in depth, and the opacity of the cornea was augmented. Perforation at last ensued, and, under the influence of pressure, issue was given to a puriform liquid. Death took place unexpectedly. On a post-mortem examination the Casserian ganglion of the right side was found to be of considerable volume and very hard. The three branches of the right trifacial were likewise found much thickened up to the point of their emergence from the bone.

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[1] G. Meissner, "Ueber die nach der Durchschneidung der Trigeminus am Auge der Kaninchens Eintretende Ernahrungstärung," 'Henle und Pfeufer's Ztsch.' (3), xxix, 96–104. 'Centralblatt,' 1867, p. 265. 'Gazette Hebdomadaire,' 1866, p. 634.

[2] Schiff, 'Henle's Zeitsch.' (3), xxix, 217–229. 'Centralblatt,' 1867, p. 655. 'Gazette Hebdom.,' 1867, p. 634.

[3] This is not the interpretation which Meissner proposed for his experiment. He supposes that the innermost fibrils of the trifacial, which had alone been cut, in the case quoted, have a special action on the nutrition of the eye. He bases his opinion on this that, in three other cases where the trifacial had undergone incomplete section, but where the innermost nerve fibrils had been respected, no trophical troubles in the eye ensued, although this organ which had lost its sensibility was not protected from external agencies. We think that incomplete sections need to be repeated a considerable number of times before it is possible to pronounce a definite judgment on the interpretation proposed by Meissner.

[4] Bock, 'Ugeskrift for Læger,' 1842, vii, p. 431. Extract in 'Hannover's Jahresbericht,' 'Muller's Archiv,' 1844, p. 47, and Schiff's 'Untersuchungen zur Physiologie des Nervenssystems mit Berücksichtigung der Pathologie,' Frankfurt am Main, 1855, pp. 63, 64.

The following case is taken from a memoir by Friedreich.[1]

A man, aged 65, was suddenly smitten by hemiplegia on the right side, with loss of sensibility on the same side. Some weeks before this attack he had felt slight lancinating pains in the left side of the face and in the globe of the left eye. These pains increased rapidly and to a high degree after the apoplectic attack. At the same time the conjunctiva of the left eye became injected, and there was an increase of the lachrymal secretion: later on, the conjunctiva was coated by a pseudo-membranous puriform exudation. The left pupil, though very much contracted, was still sensitive to the action of light. Sensibility remained normal over the whole of the left side of the face.

At the autopsy there was found at the surface of the middle peduncle of the cerebellum a collection of little sarcomatous tumours, which, taken altogether, were about the size of a filbert; the adjacent brain-substance, especially next the cerebellum, was softened and very much injected. The left trifacial nerve, at its emergence from the base of the encephalon, was red, slightly softened, and flattened by the tumour.

It would be easy to quote a considerable number of cases analogous to those we have cited, but these will suffice for the object we have now in view.[2]

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[1] Friedreich, 'Beiträge zur Lehre von den Geschwülsten, innerhalb der Schaedelhohle,' Wurzburg, 1843, p. 15, and Schiff's 'Untersuchungen,' etc., p. 100.

[2] Facts relating to nutritive disorders of the eye, consecutive on spontaneous lesions of the fifth pair in man, are numerous enough, but we have only wished to mention those in which it was well established that the facial sensibility had not been touched. The two following cases, however, are also deserving of notice, although they are not so explicit, in this respect, as those of Bock and Friedreich.

A vigorous individual, after a blow received upon the head, became subject to violent fixed pains, on the right side of the head, and suffered occasionally from epileptic fits. Afterwards, the pains became localized in the right eye and ear. The eye was red, tumefied, and projecting, but still covered by the paralyzed upper eyelid. Turbid cornea; iris, contracted and motionless; brown-coloured at first, then greenish. The cornea became, at length, opaque.

Post-mortem.—The lower surface of the anterior and middle lobes present, on the right side, several steatomata of the size of a bean, or almond. The Casserian ganglion and the three branches of the trifacial are covered over by a stiff cartilaginous mass The motor oculi is compressed, and its colour altered. The state of the sensibility of the skin of the face is unfortunately not given in this case. F. A. Landmann, 'Commentatio pathologico-atomica exhibens morbum cerebri oculique singularem;' in-4°, Leipzig, 1820, and Schiff's 'Untersuch.,' p. 51.

In the well-known case recorded by Serres, 'Journal de Physiologie,' v, 1825, pp. 223, and 'Anatomie comparée du Cerveau,' in spite of the profound alteration

Apart from the fifth pair, experimental lesions of other nerves are still more rarely found to determine the appearance of trophical troubles in the peripheral parts. We should quote, however, as examples of this species, the remarkable effects produced upon the nutrition of the kidneys by lesions of the nerves supplying them. Amongst the experimenters some, like Krimer, Brachet, Muller and Peipers, A. Moreau, and Wittich, assert that they can, almost with certainty, produce, by means of these lesions, more or less deep-seated alterations in the kidneys. Others, however, such as Paul Bert and Hermann, on repeating the same experiments under apparently identical conditions, declare that they obtained nothing but negative results.

May we not, at least partially, account for this singular contradiction in the following manner? No renal lesion was manifested when nerve-sections had been complete and thorough; on the contrary, such lesion appeared, or, perhaps I should say, may have appeared when the section was imperfect, or when the scalpel was replaced by caustics, by ammonia for instance (Corrente, Schiff), these being circumstances eminently proper to determine in the injured nerves a more or less active irritation or even to set up manifest inflammatory action.[1] From this point of view the question would probably deserve to be revised with the help of new researches.

We mentioned, a little time ago, the effects of transverse sections and partial destruction of the spinal cord in so far as regards the nutrition of parts deprived of feeling and motion in consequence of such operations. We said that, when the operations did not give rise to inflammatory action in the injured cord (and this takes place in the great majority of cases), there are found in the paralyzed members simply a degeneration with atrophy of the muscles, very slowly supervening, ulcerations of the derm, and perhaps eschars caused by dragging over a rough surface, or by continuous contact with decomposed urine, and want of cleanliness. In one word, all the effects, to which functional inertia of the hinder member of animals gives rise, are present, and these only. But the scene changes completely if, in consequence of circumstances that cannot be foreseen, nor as yet produced at will, inflammation is set

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of the Casserian ganglion and of the roots of the larger fasciculus of the trifacial, there was not complete paralysis of the sensitive portion of the nerve, for the whole surface of the face had preserved the sense of feeling. There had been acute inflammation of the right eye, with œdema of the lids, obnubilation and subsequently complete opacity of the cornea. The right Casserian ganglion was of a grayish-yellow, tumefied, and moist with a serous exudation. That portion of the ganglion where the ophthalmic nerve arises was red and injected. The roots of the larger fasciculus presented a dirty hue, contrasting with the colour of the lesser branch, which had remained healthy. The three nerves, on issuing from the ganglion, were of a yellow colour, which disappeared at their exit from the cranium.

[1] See 'Zeitschrift für ration. Med.,' 35 Bd., p. 343.

up in the vicinity of the spinal lesion. Then, indeed, as M. Brown-Séquard has shown, and as I have had many opportunities of observing, muscular change takes place with great rapidity; in but a few days after the operation, the alteration is very manifest. The emaciation of the muscular masses soon becomes appreciable, and makes very quick progress. Eruptions appear on the skin which promptly issue in the formation of ulcerations and of eschars, though the most minute care be taken to preserve cleanliness. They develop especially in those regions which are subject to pressure, to friction, to prolonged contact with the urine; but they may also make their appearance apart from these conditions, although the cases are rare.[1]

I might dwell at length on these trophic troubles connected with traumatic inflammation of the spinal cord in animals, but it will be more appropriate to recur to the matter when studying the features of myelitis, spontaneously developed in man. Besides, I have no desire to prolong overmuch this incursion into the field of experimental physiology. We have already obtained one result, if I mistake not; the facts quoted suffice, it seems to me, to establish that the *abolition of the action of the nervous system* has no direct immediate influence on the peripheral parts—on the other hand, they make it appear at least extremely probable that *morbid excitation or irritation* of the nerves or nerve-centres is of a nature to give rise to the most various nutritive derangements in distant parts.

By what means or mechanism does this irritation of the nervous system react upon the peripheral parts, and determine there lesions of nutrition such as those we have mentioned? Are these due to an irritation of the vaso-motor nerves or to their paralysis? Or do they depend on an irritation of those hypothetical nerves, which anatomy as yet knows not, and which are sometimes called *trophic nerves?* These are questions which we shall have to discuss hereafter; at present we must return to the domain of human pathology, and I hope to make you acknowledge that the principle already set out by experimental physiology has its application here in a still more manifest manner. This principle shall be our clue, and it will lead us, I hope, to comprehend why lesions, which appear at first sight similar and which are referable to the same points of the nervous or peripheral systems, should produce, in pathological cases, results so opposite and even so contradictory in appearance.

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[1] It is doubtless in the same manner, that is, by invoking the existence of inflammation about the injured point, that we should explain the disorders that sometimes occur in the nutrition of the eye, in many animals, after section of a lateral half of the spinal cord, in the back. The affections of the eye (ulcerations, melting away of the cornea, purulent conjunctivitis), observed by M. Brown-Séquard in the guinea-pig ('Comptes-Rendus de la Société de Biologie,' t. ii, 1850, p. 134), have been met with by M. Vulpian, in the frog, after the section of the corresponding half of the cord, near the medulla oblongata (this fact was orally communicated). Such affections do not supervene in all animals which have been thus operated on, and it is at least highly probable that they arise only in cases where, consequent on the section, an inflammatory action was set up in the superior segment of the spinal cord.

The trophic troubles which we purpose to pass in review are produced:—

1. By lesions of the peripheral nerves, which lesions may be either due to traumatic causes or spontaneously developed.
2. By lesions of the spinal cord and of the medulla oblongata.
3. By lesions of certain portions of the encephalon.

TROPHIC DISORDERS CONSEQUENT ON LESIONS OF THE NERVES.

Let us commence by considering lesions of the nerves. They present the simplest conditions of study. Surgery, in this respect, supplies us with records of great value; for traumatic lesions of the nerves are occasionally to be observed in the human organism, under conditions of simplicity comparable with those accompanying the experimental lesions inflicted on animals.

A. At the outset, I shall establish what I consider to be a fundamental distinction between these traumatic lesions of nerves, the importance of which you will soon perceive. 1. The lesion sometimes consists of a clean and complete section, in which case the effects are merely, speaking generally, those resulting from the absence of nerve-action; 2, sometimes, owing to contused or lacerated wounds, the lesion is of such a nature as to set up irritation in the nerve, in which case, and then only, we see arise those trophical troubles to which I invite your attention. Let us first consider the cases belonging to the second group.

The traumatic nerve-lesions in question may give rise to morbid phenomena affecting the skin, the subcutaneous cellular tissue, the muscles, the joints, and the bones. You are aware that the last American war has furnished occasion for some very important studies on this subject; they have been given to the public by Drs. Weir Mitchell, Morehouse, and Keen in an interesting work of which we shall often avail ourselves.[1] To one of my former students, the late lamented Mougeot, we also owe a very remarkable treatise on cutaneous affections developed under the influence of lesions of the peripheral nerves. It is not to be expected that I can enter into details, and consequently I must refer any of you who desire to investigate this question fully, to the thesis of Mougeot, in which all records relative to the subject have been most carefully brought together.[2]

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[1] S. Weir Mitchell, G. R. Morehouse, and W. W. Keen, 'Gunshot Wounds and other Injuries of Nerves.' Philadelphia, 1864. Extract in 'Archives Générales de Médecine,' 1865, t. i. This work has been translated into French by Dr. Dastre (1874).

[2] J. B. A. Mougeot, 'Recherches sur quelques troubles de nutrition consecutives aux affections des nerfs,' Paris, 1867.

a. Skin affections.—The accidents which traumatic nerve-lesions may occasion in the integuments are of several kinds:—

1. The first includes eruptions of various forms, but chiefly those characterized by vesicles and bullæ. We shall cite, in the first place, the *zona* (*herpes zoster*), which is frequently observed in such cases, and which on that account might be designated *zona traumatica.* I described, at the time, a very fine specimen of this kind, observed at the hospital of La Charité, under the treatment of my master, Rayer.[1] The American surgeons, already mentioned, have described, under the name of *eczematous* eruptions, an affection of the skin which may be placed with this form.

2. The second kind includes pemphigoid eruptions, of which I have also described a well-marked specimen. Here we see the pemphigus bullæ developing with great rapidity, and reappearing from time to time on different parts of the tegumentary system supplied by the wounded nerve. They leave after them well-nigh indelible scars. This kind of eruption is sometimes seen on vicious cicatrices, and is then most probably due to irritation set up in some nerve-filament which has been either strained or compressed in the cicatricial tissue.

3. In the third place we may note a cutaneous redness, which resembles *erythema pernio*;[2] and a certain tumefaction of the skin and subcutaneous cellular tissue, as remarked by Hamilton, which simulate phlegmon (pseudo-phlegmon).[3]

4. Finally, we have that cutaneous affection which has been described by the American surgeons under the name of "Glossy

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[1] "A patient, admitted into Dr. Rayer's wards in 1851, had during the troubles of June, 1848, received a bullet in the outer inferior part of the thigh. Some time after the wound had healed, acute pains in the leg supervened, which were almost continuous, but with occasional exacerbations. These pains, which seemed to start from the cicatrix, extended to the dorsum of the foot, and followed evidently the course of the nerves. This neuralgia, which resisted all means of cure employed, was several times, during the patient's sojourn at La Charité, accompanied by an eruption of herpetic vesicles, arranged in groups closely resembling those of herpes zoster and occupying the skin of the painful parts." Charcot, "Sur quelques cas d'affection de la peau, dependant d'une influence du système nerveux," 'Journal de Physiologie,' t. ii, No. 5, Janvier, 1859.

In the same Journal, an analogous case was recorded by M. Rouget. "A husbandman, whilst leaping a trench, received a charge of rabbit-shot from his gun, in the middle region and on the inner side of the left arm. In the bottom of the wound, which was over three inches wide, one saw the brachial artery, the torn basilic vein and several contused nerves, especially the internal brachial cutaneous. The wound healed quickly enough, but in about from two and a half to three months, there supervened a herpes-like eruption on the posterior-internal part of the forearm, occupying an area of some two inches in diameter, in a portion of the forearm deprived of feeling. Examples of herpes following on a contusion over the course of a nerve (Oppolzer), or resulting from a strain or an effort (Thomas), are far from rare." (*Vide* Mougeot, *loc. cit.*, p. 38.)

[2] Charcot, *loc. cit.*, 'Eruption particulière siegeant sur la face dorsale d'une main et des doigts, et probablement consécutive à la lésion des filets nerveux qui se distribuent à ces parties.'

[3] Mougeot, *loc. cit.*, p. 30.

Skin." The skin becomes smooth, pale, and bloodless; the sudoriparous glands are atrophied, and their secretion is diminished; the epidermis is cracked, and the nails likewise are cracked and curved in a very remarkable manner. Here, in fact, we have to deal with a peculiar inflammation of the skin, which recalls some of the features of the disease known as scleroderma.

b. Affections of the Muscles.—The muscles waste away often in a very rapid manner, and lose sometimes partially, sometimes completely, their electrical contractility. But this muscular atrophy will form the subject of a special study.

c. Affections of the Joints.—Traumatic nerve-lesions produce, in relation to the joints, symptoms which recall in a marked manner the features of subacute, articular rheumatism. These arthropathies usually terminate in anchylosis.

d. Affections of the Bones.—Under similar circumstances we occasionally find periostitis produced, often followed by necrosis.

But I shall not proceed further with this brief enumeration, as what we have seen will suffice for our purpose. It is important, especially at present, to endeavour to specify, as exactly as possible, the particular conditions under the influence of which these trophical troubles are developed, after traumatic lesions of the nerves.

Paget, who was one of the first to call attention to these accidents, does not hesitate to confess his ignorance in relation to them.[1] The American surgeons, on the other hand, whom we have already quoted, have succeeded in determining the conditions in question, and their testimony is the more valuable to us here, because it is based upon observation alone, wholly empirical, and free from any preconceived idea. After having remarked, as indeed Paget had done before them, that these consecutive affections are almost always preceded or accompanied by burning pains (evidently correlated to an irritative condition of the injured nerve), whilst anæsthesia is almost altogether absent, they explicitly point out that these disorders usually take place after *contusions, punctures, incomplete sections of the nerves*,—that is to say, after traumatic causes which are most competent to produce neuritis, or at least the *neuralgic condition.* On the other hand, and upon this our authors insist, these derangements are not observed to follow *complete sections of the nerves*, the common consequences of the abolition of nervous action being the only phenomena perceptible in such cases.

It should be added, in conclusion, that the peripheral affections which are attributable to nervous irritation, occur spontaneously in the majority of cases, without the intervention of pressure, or of any external cause whatever.[2]

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[1] Paget, 'Medical Times and Gazette,' London, March 26, 1864.

[2] 'Gunshot Wounds,' etc., *loc. cit.*, pp. 71–77, and 'Archives Générales de Médecine,' t. i, 1865, pp. 188, 191, 194.

But these can only be looked upon as very general conditions: we should be able to penetrate more deeply and seek whether there does not exist in the affected nerves a constant anatomical lesion that can be correlated with the manifestation of the peripheral lesions. Unfortunately, we must confine ourselves here to pointing out a lacuna which future research will not, undoubtedly, fail soon to fill. However, the symptoms, taken as a whole, plead already in favour of the existence of a neuritis. In addition, we may appeal to the necroscopical results which have been obtained, in certain cases of organic nerve-lesions, in which the whole series of peripheral affections that we have learned to recognize as a consequence of traumatic lesions may be observed. In fact, in these cases (which will shortly engage our attention), the affected nerves have been sometimes found tumefied, infiltrated with exuded matter, and greatly congested. Examined under the microscope, we discern a more or less well-marked multiplication of the nuclei of the tube-sheaths (sheaths of Schwann) or of those of the neurilemma and sometimes, moreover, all the signs of granular degeneration of the medullary cylinders. Nothing as yet, however, proves that an irritation capable of determining the production of remote trophic troubles may not exist in the nerve without being betrayed by such comparatively coarse lesions. It is opportune here to point out that every neuritis does not necessarily entail the manifestation of trophic troubles. The case is quite otherwise. In order that these should follow, the intervention of certain circumstances is required, which analysis has not as yet enabled us to describe. The fact mentioned stands out in contrast with what we know of those lesions which supervene, in distant parts, after complete nerve-sections, for the latter may be looked upon as obligatory and inevitable consequences of every nerve lesion which absolutely deprives the parts in question of the influence of the nervous system.

However this be considered, the influence of the irritation of a nerve upon the development of the nutritive derangement with which we are concerned, is set under a strong light and rendered manifest, as it were, by the observation of cases in which these accidents, after a temporary disappearance, are seen to be reproduced on every reappearance of the irritative cause. As an example, I will mention a well-known and often quoted case, which Paget relates on the authority of Dr. Hilton :—

A patient, under treatment in Guy's Hospital, for fracture of the inferior extremity of the radius was found to have acquired a voluminous callus which compressed the median nerve. Ulcers that proved rebellious to all curative efforts had formed, in consequence, on the skin of the thumb and of the first two fingers of the hand. By flexing the wrist so as to relax the soft parts of the palmar surface and thus to relieve the nerve from compression, it was always found possible to bring about the cure of the ulcers in a few days. But as soon as the patient tried to make use of his hand the nerve was again subjected to compression and the ulcers were observed to reappear with but little delay.[1]

B. It now remains for us to consider those trophic disorders which arise in consequence of spontaneously developed, non-traumatic, nerve lesions. As I have given you to understand, we shall here encounter the whole series of affections which we have just reviewed. This circumstance will permit me to deal briefly with the matter; it is enough to quote some typical examples, the majority of which I borrow from the rich treasury of facts accumulated in M. Mougeot's work.[2]

To demonstrate the existence of a transition I will mention, in the first place, certain cases in which an influence, not properly traumatic but yet belonging to the mechanical order, has determined an affection of the nerve. It is manifestly in this manner that trophic disorders of the eye, consequent on lesions of the trifacial, are produced, as in such cases the cause is usually found to be this, namely, that intracranial tumours, developed in the vicinity of the nerve, determine in it by compression a more or less active irritation without effecting any solution of continuity in the nerve-tubes. Cancer of the vertebral column may, as you are aware, bring on softening of the vertebræ to such an extent that the laminæ give way and the intervertebral notches are narrowed. The nerves in their transit through these foramina are consequently compressed, irritated, and sometimes become inflamed. Under such circumstances I have seen an herpetic eruption occupy, on the right side, all the cutaneous regions supplied by branches of the cervical plexus, in consequence of the compression to which the nerves that go to form it were subjected, in their exit through the spinal foramina. The cervical portion of the cord itself and the roots of the cervical nerves were healthy, as the post-mortem examination showed, but on opening the right foramina the spinal ganglia and the nerve trunks were found tumefied and of a vivid red colour. Moreover, in the ganglia as in the nerves we saw, on a microscopical examination, that there had been a vast multiplication of nuclei. On the contrary, the ganglia and nerves of the left side presented no trace of alteration.[3]

It is highly remarkable to find that an inflammation, strictly limited to the spinal ganglia and nerves, may be developed spontaneously and without the intervention of any mechanical cause, giving rise nevertheless, as Von Bärensprung has shown, to an herpetic eruption, in those cutaneous regions to which the nerves, under irritation, are distributed.[1] There are some reasons for believing that a considerable number of cases of spontaneous herpes make their appearance in consequence of a neuritis of this kind.[2] The spinal ganglia also have been found much altered, although neither the spinal cord, nor the anterior and posterior nerve-roots, nor even the intercostal nerves themselves participated in the lesion. A case in point has been recently noted by Herr E. Wagner.[3]

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[1] J. Paget, 'Lectures on Surgical Pathology,' v. i, p. 43.

[2] Mougeot, *loc. cit.*, chap. ii, "Des lesions organiques des nerfs et des troubles de nutrition consécutifs."

[3] Charcot et Cotard, "Sur un cas de zona du cou avec altération des nerfs du plexus cervical et des ganglions correspondents des racines spinales postérieures," 'Mémoires de la Société de Biologie.' Année 1865, p. 41.

An individual, aged 23, suffering from pulmonary phthisis, complained, towards the close of his existence, of an herpetic eruption occupying the region corresponding to the ninth and tenth intercostal nerves of the left side. On post-mortem examination, it was found that the bodies of the six inferior dorsal and two superior lumbar vertebræ had been attacked by caries. The dura mater in the region corresponding to these vertebræ was surrounded externally by a thick layer of caseous pus which extended to the nerve-sheaths and spinal ganglia. The dura mater itself was thickened and split into two laminæ, especially in the neighbourhood of the ninth, tenth, and eleventh dorsal nerve-roots. Although the lesions of the dura mater seemed as well marked on the right side as on the left, yet the ninth, tenth, and eleventh dorsal ganglia of the left side only were swollen and presented appreciable alterations under the microscope. In these three ganglia the nerve-cells had disappeared, and in the immediate vicinity of their vacant alveolæ were seen all the signs of abnormal proliferation in the connective tissue, carried to an advanced stage.

In many cases of chronic spinal meningitis, accompanied by thickening of the dura mater, I have myself observed the concomitant inflammation of the spinal nerves (in their passage through the meninges) give rise, not only to more or less well-marked atrophy of the muscular mass, but also to various cutaneous eruptions which generally presented the appearance either of zona or of pemphigus. In a lecture delivered at Dublin,[4] M. Brown-Séquard had previously pointed out the existence of special cutaneous eruptions on the arms in cases of spinal meningo-neuritis localized in the inferior portion of the cervical region.

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[1] Von Bärensprung, "Beiträge zur Kenntniss des Zoster," 'Archiv für Anat. und Physiolog.,' No. 4, 1865, and 'Canstatt's Jahresb.,' 1864, t. iv, p. 128.

[2] Mougeot, *loc. cit.*, p. 65.

[3] R. Th. Bahrdt, "Beiträge zur Ætiologie des Herpes Zoster," Diss. Leipzig, 1869, and E. Wagner, "Patholog. Anatomische und Klinische Beiträge zur Kenntniss der Gefässnerven." 'Archiv der Heilkunde,' 4e heft, Leipzig, 1870, p. 321.

[4] 'Quarterly Journal of Medicine,' May, 1865 [pp. 11, 12 of special edition].

Erythema, zona, muscular atrophy, and certain arthropathies have been correlated, by M. Duménil, with chronic progressive neuritis,[1] and by M. Leudet with peripheral neuritis, consecutive on asphyxia from charcoal fumes.[2]

But it is, above all, in *anæsthetic lepra* that we encounter in their full development the trophical disorders which we have studied in connection with traumatic nerve lesions. The initial morbid process here consists, as we learn from the important researches of Herr Virchow,[3] in a *leprous perineuritis* characterized by a special cell-proliferation, in the space between the nerve-tubes, which determines their slow destruction. The nerves, then, frequently present in their course a spindle-shaped swelling which may sometimes be readily recognized, during life, in regions where they lie superficial, as the ulnar nerve at the elbow, and thus assist in the diagnosis. These alterations give rise, at the outset, to symptoms of hyperæsthesia, and afterwards to those of anæsthesia.

With the exception of zona, which I have not seen mentioned anywhere, we find in these circumstances almost the whole series of the trophical lesions which we have already described: *a*, pemphigus as *pemphigus leprosus; b*, "glossy skin;" *c*, muscular atrophy; *d*, periostitis and finally necrosis. When the latter lesions attain a high degree of intensity we may have occasionally to note, you are aware, the loss of part of a member. This often happens without pain; because, when it does occur, anæsthesia is usually present (*lepra mutilans*).[4] Some have attributed these various accidents and mutilations to the effects of the anæsthesia. It should not, certainly, be regarded as the sole efficient cause; for it is not only proved that this merely facilitates the intervention of external agencies, but also that it can be relegated to a secondary position, and even eliminated altogether if we take into account the cases given by Dr. Thomson in which there was absolutely no anæsthesia.[5]

We have here been able only to pass rapidly in review the disorders of nutrition which result from irritative lesions of the peripheral nerves. In the following lectures we will return to the subject again, but our principal object will be to describe the trophical troubles which are correlated to lesions of the brain and spinal cord.

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[1] Duménil, 'Contributions pour servir à l'histoire des paralysies périphériques, specialement de la névrite.' 'Gazette Hebdomadaire,' 1868. Nos. 4, 5, 6.

[2] Leudet, 'Recherches sur les troubles des nerfs périphériques et surtout des vaso-moteurs, consecutifs a l'asphyxie par la vapeur du charbon," 'Archives Générales de Médecine,' Mai, 1865.

[3] R. Virchow, "Die Krankhaften Geschwülste," 'Nerven-Lepra,' t. ii, p. 521. 1864-5.

[4] F. Steudener, 'Beiträge zur Pathologie der Lepra Mutilans.' Mit 3 Taf. Erlangen, 1867.

[5] A. S. Thomson, 'Brit. and For. Med.-Chir. Review,' 1854, April, p. 496, quoted by H. Virchow.

LECTURE II.

NUTRITIVE DISORDERS CONSECUTIVE ON NERVE-LESIONS (*Continued*). AFFECTIONS OF THE MUSCLES. NUTRITIVE DISORDERS CONSECUTIVE ON LESIONS OF THE SPINAL CORD.

SUMMARY.—Anatomical and functional modifications occurring in muscles under the influence of lesions of the nerves supplying them. Importance of electrization as a means of diagnosis and prognosis. Researches of Dr. Duchenne (de Boulogne). Experiments: Long persistence of the electrical contractility and of normal nutrition of muscles, after the section or excision of motor or mixed nerves in the case of animals. Pathological cases: Diminution or speedy abolition of the electrical contractility, followed by rapid atrophy of the muscles in cases of rheumatic paralysis of the facial nerve, and of irritative lesions of mixed nerves, whether of traumatic or spontaneous origin. Cause of apparent contradiction between the results of experiment and the facts of pathology. Application of the researches of M. Brown-Séquard: Irritative nerve-lesions alone determine the speedy abolition of electrical contractility, followed by rapid atrophy of the muscles. Experiments of MM. Erb, Ziemssen, and O. Weiss. Contusion and ligation of nerves are irritative lesions. Difference of the results obtained in the exploration of muscles when faradization and galvanization are employed. The results of these new researches are comparable with the facts of human pathology; they do not weaken the proposition of M. Brown-Séquard.

Trophic disorders consecutive on lesions of the spinal cord. Considered with regard to their influence on the nutrition of the muscles these lesions constitute two well-defined groups.

First group: lesions of the cord having no direct influence on muscular nutrition: *a*, lesions in circumscribed spots affecting the gray substance to but a slight extent vertically, *e. g.*, partial myelitis, tumours, Pott's disease; *b*, extensive fasciculated lesions of the white posterior or antero-lateral columns, without the gray matter participating; *e. g.*, primitive or secondary sclerosis of the posterior, antero-lateral columns, etc.

Second group: lesions of the spinal cord which influence, more or less rapidly, the nutrition of the muscles: *a*, fasciculated or circumscribed lesions which affect the anterior cornua of the gray matter to a certain extent, in height; central myelitis, hæmatomyelia, etc.—*b*, irritative lesions of the large nerve-cells of the anterior cornua with or without participation on the part of the white fasciculi: infantile spinal paralysis, spinal paralysis of adults, general spinal paralysis (Duchenne de Boulogne), progressive muscular atrophy, etc. Predominant influence of lesions of the gray matter in the production of trophical troubles of the muscles. These facts can be interpreted by means of Brown-Séquard's proposition.

GENTLEMEN: In the preceding lecture, whilst sketching the history of nutritive troubles consecutive on nerve-lesions, I purposely avoided dwelling on the anatomical or functional modifications to be found in muscles under the influence of these lesions. I desired to reserve this question for a special study. In reality, as you will soon acknowledge, this is a subject surrounded by difficulties of all kinds, and is even now the object of a thousand controversies.

You are aware of the great progress which has been made in the clinical history of paralytic affections, under the influence of the labours of Dr. Duchenne (de Boulogne). But you know also, without doubt, that a considerable number of the facts discovered by this eminent pathologist seem to be in flagrant contradiction with the results obtained by physiologists in their experiments upon animals.

What is the reason of this discordance? In what direction are we to look for means of effecting a reconciliation between them? These are desiderata which I do not undertake to answer in a manner perfectly satisfactory on every point. Yet I must not recoil from before the difficulty; I am bound, at least, to examine it. To be candid, I feel some repugnance to treat a question, where the results of the electrical exploration of nerves and muscles must be continually referred to, in the presence of men who have made so profound a study of this mode of examination. But if they meet with criticism, I hope they will accord me their generous indulgence.

I.

We may say, in a general manner, that *electro-diagnosis*, if I may invent a term, announces and demonstrates (in certain pathological cases where a somewhat intense lesion of a mixed or motor nerve has taken place), the rapid and great diminution, it may be even the total disappearance of that property which is known by the name of electrical contractility. Yet, on the other hand, experiments on animals appear to show that, after lesions inflicted on nerves, the muscles preserve for a comparatively long time, even indefinitely, according to some authors, the property of contracting under the stimulus of electricity.

You will readily understand the interest which, from our point of view, belongs to the recognition and study of facts of this kind. It suffices to remind you that the enfeeblement and, *à fortiori*, the loss of electrical contractility, rapidly ensuing on the lesion of a nerve, are, as clinical observation has frequently shown, the first term of a series of phenomena which, if the physician do not intervene, almost necessarily entail, in certain cases, the more or less complete atrophy of the muscle and sometimes the total loss of its functions.

In order to set out in a clearer light the discordance to which

I have called your attention, allow me, gentlemen, briefly to recall the experimental facts in question:—

A. It was proposed, in these experiments, to seek out the modifications which take place in the properties of muscles and in their anatomical structure, after the section or excision of the nerves supplying them. Experiments abound; they have been made and repeated by MM. Longet, Schiff, Brown-Séquard, Vulpian, and it must be added that the results which they have given appear, at least as regards essential points, to be quite concordant. The following are the principal incidents of these experiments that seem to call for notice here.

When a nerve has undergone section or partial excision, its peripheral extremity begins, from the fifth to the sixth day after the operation, to undergo even in its finest ramifications a series of alterations whose ultimate consequence is the disappearance of the medullary cylinder, whilst the axial filament appears, on the contrary, to persist almost indefinitely.[1]

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[1] Professor Schiff has shown that, in cases of nerve-degeneration following on section, the axis cylinders persist, contrary to what M. Waller had asserted; he found the filaments in the nerve-fibres of nerves of mammalia, which had been cut five months previously. "We have also recognized," says M. Vulpian ('Leçons sur la Physiologie du Système Nerveux,' 1866, p. 236), "the existence of this axial filament at the end of more than six months. It appears to me very probable that it persists beyond this space."

Since the above was written, M. Ranvier ('Comptes Rendus de l'Académie des Sciences,' 1872) published the result of his researches on nerve-degeneration, and demonstrated the destruction of the axis-cylinder. Having had opportunities of examining his preparations whilst studying in his laboratory, I may be allowed to state that the continuity of the axis-cylinder was plainly shown to be broken, at intervals; it has been objected, in a German periodical, that this might be an appearance due to the reagent employed, but the allegation is invalid, seeing that the production of the result does not depend upon the use of one reagent. The following is a brief summary of M. Ranvier's conclusions. The interannular segment constitutes an histological unit. It is formed by Schwann's sheath, lined by a layer of protoplasm containing a lenticular nucleus; beneath is the medullary sheath, through which passes the axis-cylinder, which is probably enveloped in a protoplasmic layer, reflected at the annular constrictions which terminate the segment. Twenty-four hours after section of the ischiatic or pneumogastric nerve, in a rabbit, the nuclei in question are slightly swollen, and the outer protoplasmic layer becomes granular. Forty-eight hours after section, the tumefaction of the nucleus is greater, and the protoplasmic layer forms lumps that jut into the medullary matter, and give it an irregular shape. After the seventy-second hour, the nucleus is so swollen as to fill nearly the width of the tube—there the medullary matter, completely interrupted, leaves a space occupied by a mass of protoplasm sprinkled with fatty granulations, and inclosing the nucleus. The protoplasm is also swollen at different points and has driven back the medullary sheath, reducing it to a mere thread, or completely dividing it. Towards the end of the third day the axis-cylinder is cut across, opposite each nucleus. This fact explains the loss of excito-motricity in a nerve, which supervenes at about this date after section, according to Longet. From the fourth day, the degeneration goes on increasing, and on the sixth, the medullary matter is reduced to minute fragments, whilst the protoplasm which has become very abundant contains a considerable number of fatty granulations and its nuclei have multiplied. From the seventeenth to the twentieth day, the proliferation of nuclei does not actively proceed. When the nerve is examined

On the other hand, from the fourth day, that is to say, before even the lesions of the degeneration are appreciable, the nerve (according to M. Vulpian, *loc. cit.*, p. 235) is found to have already lost the faculty of being excited by different agencies, and in particular by the electrical stimulus. There is no difference of opinion in reference to this point. With respect to the muscle, it does not present, at first, any modification whatever of the electrical contractility. The decrease, and still more the utter loss of this property, if they do ensue, are never produced until after a considerable lapse of time, and very slowly. Here, again, there is no divergence of opinion. If some physiologists state they have seen the electrical contractility lessened, or even lost, at from six to twelve weeks after the section of a mixed nerve, M. Schiff has, on the contrary, under some circumstances found it perfectly preserved at the end of fourteen months.[1] The same statement holds good if the section be made of an exclusively motor nerve. M. Longet had already shown that, whilst the motricity of nerve is, as we have said, entirely abolished four days after section, the muscular irritability, in so far as the facial nerve is concerned, persists in the corresponding muscles for over three months.[2] After excision or section of the facial nerve, MM. Brown-Séquard and Martin-Magron have seen the irritability of the facial muscles survive, in the case of guinea-pig and rabbit, for nearly two years.[3] M. Vulpian has also borne testimony to precisely the same effect.[4] About the year 1847, in the laboratory of my excellent master, Martin-Magron, whilst working in a field, which my sensitiveness in reference to animal suffering soon caused me to abandon, I was able personally to observe the almost indefinite persistence of electrical contractility in the corresponding muscles, after the excision of the facial nerve.

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in transverse section, very few axis cylinders are found in the tubes on the twentieth day. These remarks refer to the *peripheral* end of the divided nerve: the process of degeneration in the *central* end is very different. The medullary matter is decomposed into fine granulations, accreted in oval clusters, the nuclei multiply and the protoplasm augments, without, however, segmenting the medullary matter, by jutting masses. On the contrary, the nuclei are flattened between the axis-cylinder and Schwann's sheath. The axis-cylinder itself persists, its connection with the nerve-centres being preserved, it energetically resists the destructive action of the nuclei and protoplasm. Hence, M. Ranvier has come to the conclusion that the alterations undergone by nerve-tubes, in the peripheral portion of a divided nerve, which are commonly called degenerative, have not this character in so far as the cellular elements of the nerve-tube are concerned, for this, on the contrary, displays phenomena of formative activity which have an opposite meaning to that of degeneration, in the language of anatomo-pathologists (G. Sigerson).

[1] Schiff, 'Lehrbuch der Physiologie des Menschen,' 1858–59, p. 18: M. Schiff asserts that he saw, in two cases, an excitability of the muscles persist for fourteen months after section of the corresponding nerves; the hypoglossal nerve had been divided in one case, and the ischiatic in the other.

[2] Longet, 'Anatomie et Physiologie du Système Nerveux,' t. i, p. 63, 1842.

[3] Brown-Séquard, 'Bulletins de la Société Philomathique,' 1847, pp. 74 et 88, 'Bulletins de la Société de Biologie,' t. iii, 1851, p. 101.

[4] Vulpian, *loc. cit.*, p. 235.

The result is so palpable, striking, and easy of observation that most physiologists have come, if I mistake not, to question whether muscular irritability ever disappears completely in consequence of the section or excision of the nerves. They concede, at most, that in such a case there may be produced, in the course of time, a less or greater degree of enfeeblement of the contractile property of the muscles. Almost all of them point out that if the electrical stimulation sometimes becomes powerless to determine contraction of the muscles, yet this will be produced under the influence of mechanical irritation.

It was to be supposed that the nutritive or trophical modifications corresponding to these functional changes would likewise be very slowly produced and but slightly apparent. This, in fact, is what seems to take place. Most authors appear to agree in recognizing that the atrophy of the muscle and its histological degeneration supervene only when a very long time has elapsed after the nerve section. According to M. Longet,[1] when, three months after division of the facial nerve, a post-mortem examination was made, scarcely any traces of atrophy, however slight, were to be observed. But he doubtless refers to an examination made with the naked eye. M. Schiff asserts that, when the paralysis consecutive on a nerve-section is of old standing, the muscles show a certain amount of wasting. It is probable that a certain number of muscular fasciculi waste away and disappear. In most cases, the microscope reveals that a considerable number of these fasciculi undergo fatty degeneration and present an accumulation of fat in their interspaces.[2] M. Vulpian's researches have given analogous results, but, according to this author, the fatty degeneration of the muscular fibre is often completely absent.[3]

Before comparing the facts of pathology with the results of experiments on animals, it is important to clearly understand the conditions under which the latter have been conducted. In the first place, the physiologist makes a section or excision of the muscular nerves; in the second place, he resorts to direct electrical stimulation, applied to the denuded nerve or muscle. He makes use of galvanism, almost exclusively, as an agent of exploration, and takes no heed of the difference which may exist, as to their action on nerve-fibre or muscular fascicle, between the excitation obtained by means of induction (or interrupted) currents, and that determined by the galvanic (or continued) current. These are circumstances which it is important to note, especially in relation to the experiments which I shall call old, although they do not yet date from a very remote period. We shall see afterwards that quite recent researches, in which the action of both currents has been comparatively studied, have yielded results apparently different in some respects from those furnished by former experiments.

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[1] Longet, *loc. cit.*, p. 63.

[2] Schiff, *loc. cit.*, p. 175.

[3] Vulpian, *loc. cit.*, in cases of paralysis, consecutive on nerve-section: besides the atrophy of primitive nerve-fibres which is produced in the course of time, M. Vulpian has long since remarked the proliferation of the nuclei of the sarcolemma, and some other indicia of inflammatory action. This is a very interesting fact, since noted by other observers, and one to which we shall have occasion again to refer (see note [1] *infrà*, pp. 29–30).

B. Let us now take the field of human pathology into consideration. The facts which it offers us are connected with lesions of mixed or motor nerves which either take place spontaneously or supervene in consequence of a wound.

In the first place, we will describe the phenomena observed in cases of peripheral paralysis of the facial nerve and, particularly, where that paralysis is due to the influence of cold (rheumatismal paralysis *a frigore*). M. Duchenne (de Boulogne) has shown, as you are aware, that in such a case, the electrical contractility of the muscles of the face is remarkably lessened and even appears sometimes to be extinct,[1] before the end of the first week. You will remark that between this period of seven days, which according to M. Duchenne may mark the beginning of the decrease of electrical contractility in rheumatismal paralysis of the facial nerve, and the term assigned by some physiologists for the persistence of the same property in animals, after nerve-section, the difference is great. Nevertheless, researches again and again renewed have demonstrated the perfect accuracy of M. Duchenne's assertion. Quite recently also, in a case of rheumatismal paralysis of the facial nerve, Dr. Erb, having had an opportunity of following the course of the symptoms from day to day, noted that, on the ninth day, the electrical contractility had already diminished to a considerable extent.[2] In a similar case, recorded by Dr. Onimus when, eight days after the invasion of the disease, the induction current was resorted to, the paralyzed muscles did not present the slightest contraction.[3]

The same fact is usually remarked in cases of peripheral paralysis of the facial nerve, resulting from causes other than the influence of cold, and likewise in those of traumatic paralysis of nerves of extremities. The last-mentioned generally follows, as you know, on abrupt compression, contusion, or concussion of a mixed nerve, as a consequence, for instance, of scapulo-humeral luxations. The electrical contractility has been many times found very notably lessened from the tenth, and even from the fifth day in the muscles, struck with paralysis, after such and similar accidents.[4]

Clinical observation demonstrates, you must know, that, as a general rule, the muscles which thus present a prompt diminution and, above all, a prompt disappearance of electrical contractility are soon affected by atrophy which sometimes becomes very rapidly manifest, especially in the case of paralyzed limbs. It would be highly interesting to study, in the several phases of their development, the histological alterations to which this rapid wasting of muscular masses is assignable; but this is a subject in relation to which we possess as yet but little exact information. It seems, however, to follow from some researches and particularly from a case recorded in detail by Dr. Erb, that these lesions have nothing in common with passive fatty degeneration, pure and simple, such as we find in muscles that have been long condemned to inactivity. They appear on the contrary to present the clearest characteristics of an inflammatory process, to wit, a more or less marked hyperplasia of the interstitial connective tissue, recalling to some extent what we see in cirrhosis, and a multiplication of the nuclei of the sarcolemma. Concurrently with the development of these alterations, the muscular fibres undergo a very evident decrease in their transverse diameter, but they preserve, in most instances, their striated appearance. The fatty granular degeneration of the muscular fibres is rarely met with, in these cases, and appears to be altogether accidental.[1]

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[1] Duchenne (de Boulogne), 'Electrisation localisée,' 2e édition, 1861, p. 669.

[2] W. Erb, 'Zur Pathologie und Pathologischen Anatomie Peripherischer Paralysen,' in 'Deutsch. Archiv,' t. iv, 1868, p. 539. Gradolf's case.

[3] 'Gazette des Hôpitaux,' 30 Juin, 1870, p. 298.

[4] Duchenne de Boulogne, *loc. cit.* Obs., p. 191, Paralysie suite de luxation scapulo-huméral. Obs., p. 193, Paralysie suite de contusion du nerf cubital.

[1] The following is an abridgment of the observation recorded by Dr. Erb in his interesting memoir:—

Peter Schmieg, aged 22 years, suffers from pulmonary phthisis in its last stage, and likewise has caries of the petrous portion and mastoid process. An abscess has burst in the vicinity of the latter. On the 22d of March, 1867, almost complete paralysis of the left facial nerve occurs. The paralysis is particularly evident in the frontal muscle. Investigation of the electrical contractility having been made, first on March 24th (the second day of the disorder), next on the 3d April (12th day), by means of faradization, it was found normal on both dates. On the 17th April (26th day), for the first time, it is ascertained that the frontal and zygomatic muscles of the left side contract but feebly under faradaic stimulation. On the 30th April (39th day), faradization no longer causes contraction in these muscles, the other muscles of the left side of the face respond but feebly to the stimulus. Death supervenes May 2d (40th day).

Post-mortem.—The trunk of the facial nerve bounds an abscess which has opened behind the ear: it is denuded to a certain extent. The nerve-trunk is enveloped on every side by a mass of indurated connective tissue. This connective envelope closely adheres to the external nerve-sheath of the nerve, which, however, is still free within it. To the naked eye, the branches of the facial nerve offer no perceptible modifications; on the other hand, the left frontal muscle is pale, flabby, and thin. Where the nerve-trunk is enveloped by the mass of connective tissue, there is found, interposed between the nerve-fibres, a quantity of fibrous connective tissue, with numerous oval nuclei, faintly granular. A certain number of the nerve-fibres themselves present the several stages of fatty degeneration. Many of the fibres have preserved their normal character. Some of the filaments supplying the frontal muscle scarcely show any but degenerated nerve-fibres; other filaments belonging apparently to the trifacial have all their fibres in a normal condition. The left frontal muscle is greatly altered: thick septa of newly formed connective tissue are observed, interposed between the primitive muscular fasciculi. The latter are much reduced in bulk, and contain a large quantity of nuclei. On most of the atrophied fibrils the striation

It is clear that if, in the muscular atrophy which physiologists obtain, in the long run, by section or excision of the nerve, the histological lesion were always fatty degeneration without trace of irritative action, the contrast would be extremely manifest. But unfortunately for the simplicity of things, we see that this, perhaps, is not the case.[1]

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remains distinct; on others it is scarcely perceptible. A certain number of the primitive (ultimate) bundles present the characteristics of waxy alteration, but none of fatty granular degeneration (W. Erb, *loc. cit.*, 'Deutsch. Archiv,' Bd. 5, p. 44, 1866).

[1] We reserve the right of returning upon this delicate point in the course of our lectures. At present, let it suffice to note that irritative lesions of muscles quite similar to those which have been described, have been recently recorded by very competent observers, in several animals, after section and excision of mixed or purely motor nerves, that is to say, outside the conditions which commonly cause irritative nerve-lesions. Thus, when he had cut out a portion of the ischiatic nerve, Dr. Mantegazza ('Histologisch-Veranderungen nach der Nervendurchschneidung,' in 'Schmidt's Jahresb.,' p. 148, t. 136, 1857, and 'Gaz. Lomb.,' p. 18, 1867), found, after the thirtieth day, the muscles pale, the connective tissue interposed between the primitive (or ultimate) fasciculi evidently hypertrophied, the fasciculi (themselves diminished in bulk) presenting a manifest multiplication of the nuclei of their sarcolemma, but still preserving their transversal striæ. A considerable number of the fasciculi showed a granular aspect, but the granulations dissolved in acetic acid. Professor Vulpian has, likewise, met with identical alterations in the lingual muscles of a dog, fifty days after the avulsion of the central part of the hypoglossal nerve ('Archives de Physiologie,' t. ii, p. 577, 1869). The absence of fatty degeneration in the primitive fasciculi, the atrophy of these fibres with persistence of the transversal striation, and the proliferation of the sarcolemma-nuclei, have also been observed by M. Vulpian (*loc. cit.*, p. 559) in the muscles of the human leg, in a case of resection of a segment of the ischiatic nerve dating from five months before. That being the case, we are led to admit that complete sections, excisions, and avulsions of nerves do occasionally determine irritative lesions in them: or else—if further observations should prove the fact recorded by MM. Mantegazza and Vulpian to be constant—that the muscular lesions which follow passive lesions of motor or mixed nerves are not essentially different, histologically considered, from those which supervene on irritative lesions of those nerves. If the facts should support the second hypothesis there would still be occasion, we think, to differentiate, in spite of so many analogies, between muscular alterations connected with functional inertia and those consequent on nerve-irritation. It appears, in fact, to be demonstrated that the latter supervene with much greater rapidity, and are preceded and accompanied by more or less marked modifications of electrical contractility, which do not show themselves in the former with the same characteristics, and only make their appearance at the end of a comparatively very long lapse of time.

It is to be desired that a series of researches were instituted with the special object of elucidating the question which has just been raised. There does, indeed, already exist a certain number of facts tending to demonstrate that immobilization may, by itself alone, and outside of all nervous influence, provoke in certain organs and tissues, all the characteristics of inflammatory action. I shall cite but one example: we know the articular affections, described by MM. Tessier and Bonnet, which supervene when limbs are condemned to immobility, as the treatment of certain cases of fracture requires. Quite recently, M. Menzel undertook a series of experiments, which consisted in immobilizing, by plaster of Paris bandages, a certain number of articulations of dogs and rabbits. Now, from the fifteenth day, the synovial membrane was found to be vividly injected and tumefied, the articular cavity contained red corpuscles, white corpuscles, and epithelial cells; finally, the cells of the diarthrodial cartilage were found to be

It follows, in short, from the parallel which we have placed before you, that clinical facts, though most carefully observed, are, or at least appear to be, in formal opposition to experimental facts, likewise collected by the strictest methods. We should endeavour to penetrate the reason of this discordance. Let us first seek if it can be found in the difference between the conditions of observation in which the physiologist and the physician take their stand.

The first thing which requires to be distinctly brought out relates to the mode of exploration. The pathologist finds himself forbidden to explore the muscle, except through the skin, whilst the physiologist, as we have already remarked, can act under more favourable conditions, since he may apply the electrodes directly to the nerve or muscle. It might be anticipated that, where a certain degree of diminution of the electrical contractility obtains, direct application would be still capable of determining contractions, when (indirect) exploration through the skin would, perhaps, be powerless, or would at most provoke very feeble contractions. Experiments have justified the accuracy of this anticipation. Thus in a case of club-foot, with fatty degeneration of the muscles, Valentin, after the operation, remarked contractions, of a feeble character indeed, take place in one of the most thoroughly altered muscles, under the influence of direct stimulation.[1] In this case, if we may judge by analogy, exploration through the skin would

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the seat of well-marked proliferation ('Gazette Médicale de Strasbourg,' No. 5, 1871). These researches deserve to be followed out and applied to the study of the modifications which may affect different parts of a limb under the influence of functional inertia, continued for a less or greater length of time.

[1] Valentin, 'Versuch. einer Physiologischen Pathologie der Nerven.' Leipzic und Heidelberg, 1864, 2e abtheil, p. 42.

An experiment of great interest, from this point of view, was made by my (late) master, Dr. Duchenne (de Boulogne), to whom the priority of investigation seems to belong. He relates ('Electrisation localisée,' p. 40), that having found a portion of the vastus externus denuded in the case of a wounded patient (Salle St. Bernard, Hôtel Dieu), he applied a dry electrode to the denuded portion of the muscle. The contraction which followed was accompanied by a dull sensation, characteristic of electro-muscular contraction. He next placed the same electrodes on the unharmed skin over the same muscle, and only produced a burning sensation, without muscular contraction. This result, consequently, gives emphatic support so far to the statement in the above lecture. But Dr. Duchenne (de Boulogne) proceeds to observe that having replaced the metallic electrodes by moist sponges, inclosed in excitator-cylinders, and placed these on the skin, he obtained contraction with the same dull characteristic sensation, previously produced by application of the metallic electrodes to the denuded muscle. As this experiment was not made with a view to determine the exact difference caused by the interposition of the skin, the latter statement does not conflict with any given in the text; whilst the circumstances of the two experiments amply justify the view taken by Professor Charcot. It is logical to suppose that the difference of electrical action, due to the interposition of the skin, which was noted in the first experiment, would still remain, though to a very much less extent, in the second experiment, and would have been probably detected by Dr. Duchenne (de Boulogne) had his investigation been conducted with the object of ascertaining the precise amount of obstruction given by the skin. The experiment of Valentin helps to fulfil this desideratum (S.).

probably have given no result. Some facts, borrowed from the domain of experimental physiology, furnish similar evidence.

When, in the case of a rabbit whose right facial nerve had been cut across about a month before, electricity was applied through the shaven and moistened skin, to the right facial muscles, no apparent effect was produced, whilst extremely strong contractions were caused, when the homologous region of the left side was acted on. But, when the muscles were denuded on the right side, where the nerve was cut, and electricity applied to them, very marked contractions were produced. Again, a segment of the left external popliteal nerve, about two inches in length, was excised, the animal, this time, being a vigorous horse. One month after the operation, the hair was shaven off the antero-external surface of each leg, and the electrodes of a pile were applied. On the healthy leg energetic contractions followed the application; but no contraction was caused in the other, when the same test was attempted. Then, the paralyzed muscles were denuded and the stimulus was applied to them directly, the minimum power of the instrument being employed. Lively contractions were the consequence.[1] A considerable number of similar examples could, doubtless, be collected, without difficulty. Hence, it is demonstrated that exploration through the skin can only supply approximative data, and that it does not reveal the real state of the electrical contractility, but such as they are, the data furnished are not the less exact and must be considered as of the highest importance, for it is impossible not to acknowledge that the great decrease or apparent loss of contractility, detected by exploration through the skin, corresponds to a diminution or, at least, to a very great modification of this property.

Another question which requires to be considered relates to the nature of the electrical agency employed in exploration. Galvanization, as I mentioned just now, has been almost the only means made use of in the experiments concerning nerve-sections in animals, whilst in clinical practice, following Dr. Duchenne's method, the work of investigation has been pursued until lately, by means of faradization, exclusively. Now, it follows from researches made a few years ago in Germany, and recently taken up in France, that galvanization has often power to cause muscular contractions even where faradization seemed to indicate an absolute loss of electrical contractility.

This fact, recorded for the first time by Baierlacher, in 1859,[2] in a case of facial paralysis, has been remarked since, either under the same circumstances, or in different cases of paralysis consequent on traumatic lesions of mixed nerves, by Schultz,[3] Brenner,[4] Ziemssen,[5]

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[1] Experiment of M. Chauveau, in Magnin: 'Thèse de Paris,' p. 21, 1866.

[2] Baïerlacher, 'Bayz. ärztl. Intelligenzblatt,' 1869.

[3] Schultz, 'Wiener medic. Wochenschr.,' No. 27, 1860.

[4] Grünewaldt, 'Ueber die Lahmungen des Nerv. facialis.' Pet. med. Ztsch., Bd. iii, p. 321 ff., 1862.

[5] Ziemssen, 'Elektricität in der Med.,' 2 aufl., 1864.

Rosenthal,[1] Meyer,[2]—by Bruckner,[3] in pseudo-hypertrophic paralysis, and, finally, by Hammond, in infantile paralysis. From this it will be seen that galvanization may still reveal contractile power present in many cases of paralysis, whether arising from rheumatic or traumatic causes, when an exploration, conducted by faradization only, would indicate a profound alteration of electrical contractility. But, even though this is the case, the character drawn from the abolition or rapid decrease of *faradaic contractility* would subsist, not the less, in all its value; it will still allow us to maintain the contrast between paralyses from nerve-lesions, such as clinical practice usually presents, and paralyses determined in animals by nerve-section, since the character in question is deficient in the latter cases.

We have now to examine if the nerve-lesions which provoke a prompt modification of the electrical contractility, soon followed by muscular atrophy, can be assimilated without reservation, as some authors appear to believe, to nerve sections, as performed upon animals. In reality, gentlemen, this is not at all the case, and, if I err not, it is in this circumstance that we must seek the knot of the disputed question. We may say, speaking generally, that the sections or excisions of nerves do not usually set up, in these, any reactive process. The degeneration of the fibres of the distal end, which follows the operation as a necessary consequence, may be considered, supposing no complication to interfere, as a purely passive process. The muscles supplied by the divided nerves are necessarily smitten with functional inertia; but they do not appear to undergo any other changes than those which, in the course of time, result from inaction.[4]

Very different are the affections of the nerves to which are assignable, in man, the disorders that constitute the object of our study. They arise almost always when of traumatic origin, as we have said, under the influence of causes such as concussion, contusion, compression, imperfect division—all eminently calculated to provoke, in the different tissues which enter into the composition of a nerve, the development of irritative action. In fact, it is not rare, in cases of this kind, that muscular wasting of a rapid type, galloping atrophy as it were, announced almost from the outset by the diminution and loss of the faradaic contractility, should be preceded, accompanied, or followed—where a mixed nerve is concerned—by more or less acute pains, or by abnormal sensations, all being indicia of the irritation set up in the sensitive nerve-fibres.[5] To these pains may often be added the appearance of trophical skin-disorders, pemphigoid eruptions, glossy skin, herpes—which we have come to recognize as one species of the effects of irritative lesions of the cutaneous nerves, and which are never seen, in any shape or form, after simple sections of the nerve-trunks.[1] The phenomena of spontaneously developed diseases lead to identical conclusions. Sometimes caries of the petrous bone exists, the trunk of the facial nerve lies in the pus, where it is completely surrounded, as in the case which Dr. Erb records, by a dense sheath of newly formed connective tissue.[2] At other times, the nerve is compressed by a slowly developed tumour which must irritate, for some space of time, the nervous fibres before completely crushing them—nay, there is none of the series, even including so-called rheumatismal paralysis, or paralysis *a frigore* (though here we, as yet, lack positive evidence) which should not apparently be attributed to inflammation of the connective sheathing of the nerve-trunk.[3] I am not unaware that complete nerve-sections are somewhat frequently met with in surgical practice: I know also that in such cases you may see atrophy of the muscles and loss of electrical contractility supervene. But I do not believe that many cases of this kind can be adduced in which an observer has noted *from the first days diminution or loss of faradaic contractility, and from the first weeks, atrophy and degeneration of the muscles.* Although I have made some researches on this subject, I have not found, up to the present, any cases unquestionably possessing this characteristic.

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[1] Rosenthal, 'Elektrotherapie,' etc., 2 aufl., 1869.
[2] Meyer, 'Die Elektricität,' etc., 2 aufl., 1861.
[3] Brückner, 'Deutsch Klinik,' No. 30, 1865.
[4] See ante, note [1], pp. 29-30.
[5] Duchenne (de Boulogne), *loc. cit.*, Obs. ix, x.

We are thus induced, gentlemen, to have recourse here also to the luminous distinction proposed by M. Brown Séquard: *nerve-irritation alone is capable of determining rapid and early atrophy of the muscles, preceded by decrease or disappearance of faradaic contractility. Complete nerve-division does not induce atrophy and loss of electrical reaction until after an incomparably greater lapse of time, as in the case of prolonged inaction.*

That being conceded, we have now to seek in what manner—an irritative nerve-lesion being given the existence of which has been recognized—we can derive from it, as a more or less direct consequence, the rapid loss of electrical contractility, the early atrophy of the muscles, and, in a word, the whole series of phenomena which clinical observation discerns in the cases that engage our attention.

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[1] To mention one example, see the case recently reported by Dr. Constantin Paul ('Société de Thérapeutique,' Séance du 7 Mai, 1871, in 'Gazette Médicale,' p. 257, No. 25, 1871). One of the most remarkable derangements of nutrition produced by nerve-lesions is the emaciation or atrophy of the muscles supplied by these nerves. This atrophy may exist alone, or may be associated with other nutritive disorders of the same kind occupying the skin and its appendages (Mitchell, Morehouse, and Keen, 'Gunshot Wounds,' etc., p. 69).

[2] See P. Brouardel, 'Lésions du rocher, carie, nécrose, et des complications qui en sont la consequence,' Extrait du 'Bulletin de la Société Anatomique,' Paris, 1867.

[3] F. Niemeyer, 'Lehrbuch der Spec. Pathologie und Therapie,' 7e aufl., 2e Bd., p. 365.

The enfeeblement or loss of contractility is, you are aware, next to motor paralysis (which, in the vast majority of instances, heads the procession), the first fact which we take note of, in such circumstances. Some authors seem to see, in this symptom, a very simple consequence of the loss of excitability in the nerve, supervening here at an early period (about the fifth day), as in the case of nerve sections, and assignable to degeneration of the medullary sheaths, beyond the injured part. It appears certain that the contractions of the muscles, caused by electricity, are more marked when we can act upon them through the medium of the nerves, than where the stimulus, in consequence of the distinction of the nerve-filaments, can no longer bear upon the contractile substance itself. But however this may be, if the opinion to which we refer were well founded, the marked enfeeblement or apparent abolition of the electrical contractility supervening some days after the operation should be a constant fact, after nerve-sections; since in such cases the distal end of the nerve always loses its excitability at the end of five or six days. Now, we know that this is not the case. On the other hand, it is by no means proved that nerve-lesions which induce early loss of electrical contractility are always sufficiently intense to completely interrupt the continuity of the nerve-fibres and determine the destruction of the medullary cylinder. A certain number of facts might, in truth, be cited which tend to demonstrate that the continuity of the nerve persists, at least to some extent, even after lesions which rapidly occasion the appearance of the most evident trophic disorders in the muscles.

Thus, after a traumatic lesion bearing on the course of a nerve, we sometimes see the power of motion remain, and only become enfeebled when trophic lesions have supervened in the muscle.[1] It is important to note, besides, that muscular and cutaneous sensibility are often preserved, in a nearly normal condition, in cases of lesion of mixed nerves, even when rapid enfeeblement of the electrical contractility and consecutive muscular atrophy are carried very far. This is a fact to which due prominence has been given by MM. Duchenne (de Boulogne),[2] Mitchell, Morehouse, and Keen.[3]

Is it probable that, in such cases, the motor-fibres would have undergone great alterations, whilst the sensitive fibres, intermingled with them throughout the nerve, would have alone been spared? But take an argument of a somewhat more direct bearing. After certain affections of the spinal cord, such as hæmatomyelia, acute central myelitis, infantile paralysis—diseases in which the initial lesion occupies most especially the gray matter—it is common, when irritative nerve-lesions are in question, to see a diminution or a total abolition of the electrical contractility supervene in the muscles of the paralyzed member. The muscular nerves have been several times examined, in such cases, under the microscope; sometimes, they appeared normal, at others, they presented to a certain degree the changes characterizing fatty granular degeneration, but then these alterations were not at all in proportion, either in extent or in intensity, to the muscular disorders. We shall have occasion again to refer to this important fact.

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[1] See the observation quoted by Duchenne (de Boulogne), *loc. cit.*, p. 207.

[2] "In paralytic diseases consecutive on traumatic lesions of mixed nerves, the functional derangements affect the sensibility of muscles less than their contractility. Thus, a luxation of the shoulder-joint having occasioned a lesion of the nerves that supply the arm, forearm, and hand, I have known the patient to complain of rather marked muscular sensation, even when these muscles did not exhibit the slightest contraction under the influence of the most intense electrical excitation. Cutaneous sensibility is less affected than muscular sensibility, in these same nervous lesions."—Duchenne (de Boulogne), *loc. cit.*, p. 216.

[3] Mitchell, etc., *loc. cit.*, p. 97.

You see by what precedes that, in my opinion, the rapid abolition of electrical excitability observed after nerve-section cannot be altogether assigned to the fatty granular degeneration of the medullary sheath, and to the loss of excitability of the nerve fibres, which would be the consequence of this alteration. If this be so, it becomes very probable that the phenomenon in question is, at least in part, the effect of some change supervening in the constitution of the contractile substance, under the influence of the irritation transmitted to the primitive muscular fasciculus through the terminal nerve-branches. The rapidity which may mark the production of this trophic trouble is not an argument against our hypothesis. Experience, in fact, demonstrates that under the influence of certain causes, such, for example, as the abrupt interruption of the course of arterial blood, the muscular fibre may experience still more rapidly—after a few hours merely—a modification which is undoubtedly closely analogous, for it also reveals its existence by the abolition of the specific contractile power of the muscle.[1]

To judge by the usual concatenation of phenomena, discerned by clinical observation, this alteration of the contractile fibre, manifested externally by modifications of the electrical contractility, would be the precursor and the first term of a series of graver lesions which gradually lead to the wasting of the muscle, and sometimes induce the complete and definitive abolition of its functions. Observations, to which we have already referred and to which we shall hereafter return, appear to indicate that the lesions in question are largely of an irritative nature. One might be tempted after that, following the wanderings of the theory at present in vogue, to consider these lesions as the more or less direct consequence of a paralysis of the vaso-motor nerves, concomitant with the paralysis of the motor muscular nerves. Amongst the arguments which have weight against this view, we may confine ourselves to pointing out one, namely, that the requisite signs of vaso-motor paralysis—the repletion of the bloodvessels and the augmentation of the temperature—are not to be seen, except on very exceptional occasions, in those patients who, after a nerve-lesion, suffer from paralysis with rapid diminution of electrical contractility.

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[1] "I divided one ischiatic nerve in each of two rabbits and two guinea-pigs. Ten days after, I perceived that the divided nerve produced no movement when I galvanized it. The muscles contracted actively when I applied both poles to them. Having noted this, I tied the aorta below the origin of the renal arteries, and three hours after I again tried the effects of galvanism. There were no contractions set up in the leg, neither when I stimulated the nerve, nor when I directly stimulated the muscles. I then loosed the ligature; at the end of a very short time, the muscles again became irritable. The ischiatic nerve did not recover its lost property. In this experiment the muscles of the leg, after having completely lost their irritability, only recovered it by nutrition, since neither the nerve-centres nor the divided ischiatic nerve could give it."—(Brown-Séquard, 'Journal de Physiologie,' t. ii, p. 77, 1859.)

Numerous facts attest, on the contrary, that the skin is generally pale and bloodless, whilst, at the same time, the local temperature is manifestly lowered from the very beginning.[1]

II.

Such, gentlemen, was the solution of the vexed question, which I had accepted, when I became acquainted with some new investigations that had been carried out in Germany. The results furnished by these researches, where numerous experiments on animals are paralleled with pathological facts, seemed to me, at first sight, destined to destroy the whole edifice. For, indeed, to judge by the conclusions formulated by the authors, the opposition between passive and irritative lesions of the nerves, considered in reference to their effects on the contractility and the nutrition of muscles, would be anything but established. I shall set out by declaring that the experiments to which I allude, instituted by Dr. Erb, in 1868, and by MM. Ziemssen and O. Weiss, at the same period, but independently, appear to have been most carefully conducted. It remains to be seen whether they have the signification which has been attributed to them.

Various nerve-lesions—by contusion, ligature, and section in a very few cases—having been produced in rabbits, it was purposed to make daily observations of whatever modifications of electrical contractility should take place, as regarded nerves and muscles, under the action of the continued current and of faradization, each being employed in its turn. The electrization was applied sometimes through the skin, as in clinical cases, sometimes directly, as in physiological research. Dr. Erb also undertook the task of following as far as possible, from day to day, the histological alterations corresponding to the changes of electrical excitability.

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[1] Duchenne (de Boulogne), *loc. cit.*, p. 234. Mitchell, *loc. cit.*, p. 134. Folet, 'Etude sur la temperature des parties paralysées,' Paris, 1867, p. 7.

Let us, in the first place, examine the phenomena observed in these cases in connection with the injured *nerves*. Suppose that the ischiatic nerve of a rabbit has been injured from being crushed with a forceps. The lesion may be either slight or severe. If it be severe, an almost immediate loss of the electrical contractility is noted, whether galvanism or faradization be employed. On the regeneration of the nerve, the recovery of the excitability takes place slowly as regards the central end, but quickly, on the contrary, in the distal. If the lesion be slight, the electrical excitability returns rapidly to the central end, whilst it never completely ceased to exist in the peripheral extremity.

You see that these first results do not diverge perceptibly from those obtained in former experiments, since it was likewise shown by them that a divided nerve loses its excitability from the first days.

Let us now study the phenomena which, in the new experiments, are set prominently forth by the electrical exploration of the muscles. Here, gentlemen, the results diverge in a marked manner from those furnished by former experiments, and approximate, on the contrary, very closely to pathological facts.

Thus, *faradaic* exploration indicates, from the first days, a diminution, and afterwards—from five to fourteen days in intense cases—total abolition of the contractility.

This is not all. *Galvanic* exploration, also, detects an enfeeblement of the muscular contractions, from the first days; but, after the second week, this enfeeblement is followed by an exaltation which persists during the whole period of faradaic depression, and which disappears in its turn, when faradization resumes its power.

The muscular lesions which correspond to these modifications of electrical contractility have been studied with great care by Dr. Erb; for many reasons they deserve the name of cirrhosis of the muscles, proposed by Dr. Mantegazza.[1] They distinctly and completely recall those lesions which the first-mentioned author pointed out in a case of facial paralysis, observed in man.

The first alterations make their appearance in the interstitial connective tissue: from the first week there is an accumulation of numerous rounded cell-elements, recalling the tissue of granulation; these afterwards assume an elongated form, disappear, and give place to undulated connective tissue. The muscular fasciculi do not begin to show any alterations till about the second week. At this period, the diminution of their diameters is already noticeable. This atrophy makes rapid progress. However, the transversal striation persists, and the fibres never offer any trace of the alterations characterizing granular fatty degeneration. On the other hand, from a very early period, the nuclei of the sarcolemma are seen to multiply and group together in little collections, whilst concurrently with this the contractile matter presents different phases of those modifications of structure known as waxy degeneration.

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[1] Vide *supra*, note, p. 29.

Such are the phenomena pointed out as consecutive on nerve-lesions which, according to our authors, would be equivalent to complete sections. Well, I have no hesitation in declaring that this parallel is far from being secure from criticism. The results obtained by Dr. Erb and by Dr. Ziemssen relate to conditions, undoubtedly, comparable with those which pathology gives us, but not at all with those which were determined in the old experiments. Let us see how these observers have proceeded in the great majority of instances. It will be found that they almost always ligatured the nerve more or less tightly, or else crushed it more or less completely with a forceps. Now do not these circumstances furnish ample reasons for presuming that irritation of the nerve filament may have intervened here, as, in our opinion, it intervenes in pathological cases?

But there is no question of a mere presumption. The existence of an inflammation occupying not only the neighbourhood of the contused points, but even the whole length of the peripheral portion of the injured nerve, is placed beyond doubt by the descriptions of Dr. Erb himself. The neurilemma, especially, shows the characteristic signs of the inflammatory process; from the first week, rounded cell-elements, having a solitary nucleus, are seen accumulated in great numbers. At a more advanced period, a more or less thick layer of fibrous tissue is found interposed between the nerve-filaments which have undergone the different phases of fatty degeneration. As a consequence, the nerve-bundle has acquired a consistence which enables it to resist dilaceration more tenaciously than a normal nerve.

It seems to us rational to admit that in these experiments, as in the cases relating to man, the irritative lesions which occupy the nerves extend their influence to the muscles. It may, indeed, appear difficult to conceive that a nerve, which has undergone the alterations of fatty degeneration and is deprived of its motricity, should still possess a certain amount of vitality, and be capable, under the influence of an irritative lesion, of reacting on the muscular fibre, and determining trophic disorders therein. It is proper, however, to point out, in reference to this, that the irritation probably dates from the very moment when the nerve was ligatured or contused. It is certain, besides, that the vitality is far from being definitely destroyed in nerves completely separated from the nervous centres, since the work of regeneration can proceed without the distal and central ends being united.[1] It is useful to remember, also, that it is only on hypothetical grounds and without proof positive, that nerve-tubes deprived of their medullary matter and reduced to the axis-cylinder, are said to be denuded of every kind of vital property.

We must not forget, however, that ligature and contusion are not the only means employed in the experiments of Drs. Erb and Ziemssen. These authors have also made sections and excisions of nerves, though in comparatively few cases. They state that the results are always identical, whether complete section or contusive lesion be in question. But if we go back to the details of the observations, it is not difficult to see that this conclusion cannot be accepted, without reserve. We find in Ziemssen's work, especially, a very significant chapter, considered from this point of view. It treats of cases in which excisions of the ischiatic nerve were made, the excised portions being some millimetres in length. Now, the results obtained in consequence of such a lesion are very different from those which this author and Dr. Erb remarked after ligature and contusion of nerves. They resemble, in many respects, the facts gleaned from physiological experiments. Thus, in the first place, the electrical contractility diminishes, after excision, in a progressive manner, but very slowly. It does not seem to be abolished until many months have passed,—not disappearing at an early period, varying from the fifth to the fourteenth day, as happens in the case of contused lesions. In the second place, we do not meet here with that opposition between the effects of faradization and those of galvanization which was observed in cases of contusion, and which exists, as you remember, in most of the pathological cases where man is concerned. These two modes of exploration, on the contrary, produce precisely parallel effects: faradaic contractility and galvanic contractility grow weak together, and together resume their wonted intensity, on the regeneration of the nerve which, indeed, requires to be long waited for.[2]

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[1] Vulpian, 'Système nerveux,' *loc. cit.*, p. 269.

[2] Compare the observation No. ii, fig. 3, in the memoir of Ziemssen and Weiss (*loc. cit.*, p. 589), relative to a case of ligature of the anterior tibial nerve in a rabbit, with the observation No. ii (p. 593), concerning excision of the ischiatic, in a rabbit also. In the first case, faradaic contractility seemed extinguished from the 12th day after the operation, whilst galvanic contractility, on the other hand, increased from the 2d day, and kept to a high level until the moment when faradaic contractility approached its normal standard again (the 44th day). In the second case, on the contrary, both faradaic and galvanic contractility became enfeebled together and progressively, but in a very slow manner. They ceased to be manifest almost simultaneously about the middle of the third month only, and made their appearance together about four months and a half after their cessation. MM. Ziemssen and O. Weiss express themselves in the following terms in reference to the effects of the excision of the ischiatic in a rabbit. "In animals, on which this operation had been performed," "galvanic excitability declined progressively, and this enfeeblement was not preceded by a stage of exaltation. It proceeded slowly, at the same rate as the enfeeblement

If I do not deceive myself, we may conclude from this sketch that, when there is question of complete section or of excision of the nerves, the recent observations harmonize on all essential points with the old researches. On the other hand, the results obtained by MM. Erb and Ziemssen, from the employment of contusion and ligature of the nerves of animals, are comparable with the phenomena which occur in man in consequence of irritative lesions of mixed or purely motor nerves.

Now, if this be so, the dissidences which we pointed out at the beginning of this study are smoothed away, and consequently we have reason to acknowledge, in speaking of muscular affections, *the fundamental distinction between the effects of the absence of action, and those of morbid action of the nervous system* which we have already put prominently forward in discussing cutaneous and articular affections.[1]

TROPHIC DISORDERS CONSECUTIVE ON LESIONS OF THE SPINAL CORD.

Irritative lesions of the nervous centres, like those of the nerves, have the power of producing remote trophic disorders in different parts of the body. In the exposition of these consecutive alterations which we are going to offer you, we shall again find the whole series of morbid affections (with the exception of some slight differences) which we have remarked taking place after nerve-lesion. The knowledge of their history, which we have already gained, will singularly facilitate the task that remains to be accomplished.

We may say, in a general manner, gentlemen, that the *skin*, the *muscles*, the *joints*, the *bones*, and the *viscera*, may become the seats of various trophic disorders, consequent on lesions of the spinal cord and of the brain.

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of farado-muscular excitability. Galvanic excitability disappeared in the second half of the 3d month, to reappear towards the 7th or 8th month" (*loc. cit.*, pp. 592 et 593).

[1] Recent experiments made by M. Vulpian ('Archives de Physiologie,' t. iv, 1871–1872, pp. 757, 758), confirming on almost every point those of MM. Erb and Ziemssen, establish that the effects of section of peripheral nerves upon the physiological properties and structure of the muscles do not essentially differ from those caused by the application of various irritative agencies, to the same nerves, such as local contusion, ligature, and cautery. On the other hand, the histological observations of MM. Neumann ('Archiv f. Heilkunde,' Leipzig, 1868), Ranvier ('Comptes Rendus de l'Académie des Sciences,' 30th December, 1872), Eichhorst (Virchow's 'Archiv,' 1874, 12th December), have placed beyond doubt that in the peripheral extremities of the divided nerve alterations are constantly produced, *e. g.*, multiplication of the cells of the inter-annular segment, which betray the presence of an irritative process. The opposition between the effects of nerve-section and of nerve-irritation cannot, after this, be any longer maintained in the strict terms in which it was set out in this lecture (*Author's note to the 2d Edition*).

Let us take the affections of the *muscles*, in the first place, since the investigation we have just concluded has led us towards that question. The considerations we are about to set forth concerning these affections relate only to lesions of the spinal cord and medulla oblongata; for it is at least very doubtful whether lesions of the brain proper have ever the effect of directly producing alterations of the muscular tissue, and this, as we shall see in due time and place, is a fact of the highest importance.

Muscular lesions consecutive on affections of the spinal cord.—Of irritative spinal lesions, there are some which determine the very rapid production of all the kinds of functional and organic muscular alterations which we have been studying, as consequences of nerve-lesions. There are others, on the contrary, where the electrical contractility and the trophic condition of the muscles are preserved in perfect integrity, during a comparatively considerable lapse of time, for months and occasionally even for years. The muscle, in the latter case, only becomes slowly altered, under the influence of the functional inertia to which the limbs, stricken with motor paralysis, are subjected. Hence we find it possible to separate the irritative spinal disorders into two very distinct groups, which we shall pass successively in review.

A. In the *first group* we place those of the irritative lesions of the cord which, as a rule, do not directly modify the nutrition of the muscles. They have one character in common:—all tend to limit themselves to the white fasciculi of the cord, and if the gray matter be, at times, invaded, the region of the anterior cornua is respected, or at least the great multipolar nerve-cells which occupy that region are spared. Such are the different forms of *fasciculated sclerosis*, whether it be protopathic or consecutive on a circumscribed lesion (*en foyer*) of the brain or spinal cord, whether it occupy the posterior fasciculi only, or the lateral fasciculi only, or both simultaneously; so long as the express condition stated be fulfilled, that is to say, so long as the integrity of the great nerve-cells be preserved, the lesions in question may attain their highest degree of development, may, for instance, invade the white fasciculi in their whole width and their whole height, without direct deterioration to the nutrition of the muscles animated by nerves issuing from the injured portions of the cord.[1]

The scene would necessarily change, if the irritative process, exceeding the limits usually assigned it, happened to extend from the white fasciculi to the anterior cornua of the gray matter; then, in consequence of the participation of the motor-cells, a more or less rapid and thorough atrophy of the muscles might supervene.

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[1] Charcot et Joffroy, "Deux cas d'atrophie musculaire progressive avec lésion de la substance grise et des faisceaux antéro-latéraux de la moelle épinière," in 'Archives de Physiologie, t. ii, p. 635.

It is in this way, as I have pointed out elsewhere,[1] that the symptoms of general spinal paralysis or of progressive amyotrophy are sometimes superadded to the classic symptoms of posterior or lateral sclerosis, etc. We have quite recently seen many cases of this kind where we had opportunities of observing necroscopically, in the most distinct manner, the alteration of the nerve-cells to which, according to my view, the trophic muscular lesion should be attributed.[2]

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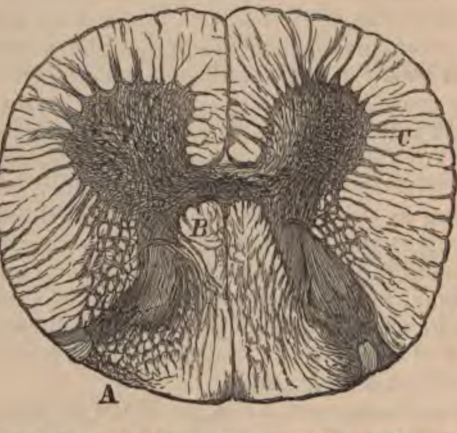
[1] Charcot et Joffroy, *loc. cit.*, p. 354.

[2] See, amongst other cases, that recently published by one of my students, M. Pierret,* 'Sur les altérations de la substance grise de la moelle epinière dans l'ataxie locomotrice considérées dans leurs rapports avec l'atrophie musculaire qui complique quelquefois cette affection, (in 'Archives de Physiologie,' etc., t. iii, p. 599). In this instance, phlegmasic action had extended from the posterior columns to the right anterior cornu of gray matter, following the course of the internal radicular fibres of the corresponding side, the consecutive muscular atrophy was exactly limited to the members of the right side (*vide* fig. 1).

The following is a succinct account of a case which shows well the mechanism by which consecutive unilateral fasciculated sclerosis may, by extending to the gray matter, determine the production of muscular atrophy :—

A woman, aged about 70 years, had been stricken with left hemiplegia, consecutive on the formation of a blood-clot in the right cerebral hemisphere. The members of the paralyzed side, which had at an early period been contractured, commenced to diminish in bulk, not quite two months after the attack. The muscular wasting affected all parts of the paralyzed members, in an uniform manner; it was accompanied by a very marked decrease of electrical contractility



* Fig. 1.—The figure is illustrative of M. Pierrett's case (a summary of which is appended); it represents a transverse section of the spinal cord taken from the lumbar enlargement. *A.* Posterior roots. *B.* Internal radicular fasciculi traversing the area of the posterior columns. The sclerosis is seen limited, in the posterior columns, to the course of these fasciculi On the right, the phlegmasic process has extended, along the course of the radicular fasciculi to the right anterior cornu, *C.* This cornu has evidently suffered diminution, in every diameter, moreover the external group of motor cells has completely disappeared, and we find in its place a dense, opaque, apparently fibroid tissue, containing numerous disseminated myelocytes.

The same rule holds good for *disseminated sclerosis* (*sclérose en plaques*),[1] and for the *diffused scleroses.* The same may be said of *primary partial myelitic affections* or of those determined by tumour-pressure, Pott's disease of the vertebræ, etc. These different diseases have no direct influence over the nutrition of the muscles so long as they do not involve the system of motor nerve-cells.

We can scarcely conceive of an exception, save in those very rare cases, where the lesion, though circumscribed to the white fasciculi, occupies that portion of the cord traversed by bundles of nerve tubes, from which issue the posterior roots. If these bundles should be at all involved in the alterations, the equivalent of a lesion affecting the peripheral nerves would necessarily be produced.[2]

B. The *second group* will include those affections of the spinal cord, the almost inevitable consequence of which it is to determine more or less grave disorders in the nutrition of the muscles. This group may be separated into two subdivisions:—

1st. The first includes those acute or subacute lesions, whether *diffuse* or *circumscribed* (*en foyer*), which involve a great length of both the white and the gray substances, but which generally predominate in the latter. They are commonly followed by great

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and made rapid progress. At the time when the atrophy became evident, the skin of the affected members, on all points subjected to the slightest pressure, presented bullæ, which soon gave place to eschars. At the autopsy, we observed, on examining hardened sections of the brain, that the descending fasciculated sclerosis of the left lateral column had been propagated to the anterior cornu of the gray matter of the corresponding side, and had there caused atrophy of a certain number of the motor-cells.

[1] In the case of a woman, suffering from multilocular cerebro-spinal sclerosis, whom we treated some years ago, one of the sclerosed patches had invaded, near the mid-cervical region, almost the whole of the gray matter of the cord, for a certain height, and the anterior cornua in particular. At this level the nerve-cells mostly presented grave atrophic lesions, and a good number of them had vanished, without leaving any trace. The woman's hands presented the deformation known as a *griffe*, *i. e.*, they were claw-like. The muscles of the thenar and hypothenar regions, as well as the interossei were atrophied; the forearms also showed great atrophy, limited to certain groups of muscles.

[2] In reference to partial myelitic affections, whether protopathic, or determined by the vicinity of a tumour, the following remark will not be out of place: They are most commonly found at a point of the dorsal region of the spinal cord, which they occupy for but a very small extent, in height. It would follow from this arrangement that if, whether primarily or in consequence of concentric extension of the morbid process, the anterior cornua of the gray matter became involved, the muscular lesions which result therefrom would be confined to very circumscribed regions of the thorax or even of the abdomen, and might not betray themselves during life, by any perceptible symptom. At all times, the nutrition of the muscles of the extremities, if there be no complication, remains perfectly intact, when the partial myelitis occupies the position we have just mentioned. It would be quite otherwise in any case where a focus of myelitis, even though very much circumscribed, should occupy certain parts of the cervical or lumbar enlargements. The muscular lesions supervening, in consequence of the invasion of the anterior cornua, would then have their seat in the extremities and would betray their presence by functional disorders, and by alterations of form, in those parts, which could not long escape attention.

modifications of electrical contractility, and by a rapidly developed atrophy of muscular fibre.

I will refer to *acute central myelitis*, in the first place. When it has been somewhat generalized, and occupies, for instance, a considerable portion of the dorso-lumbar swelling, the early diminution of electrical contractility in the lower extremities is a symptom which probably is never completely absent. Herr Mannkopf has seen, in such cases, the electrical contractility remarkably altered seven days after the appearance of the first symptoms.[1] When the patients are not too quickly carried off, you may follow the development of the correlated phenomena—the atrophy of the muscular masses soon shows itself, and the histological lesions of the primitive (ultimate) fasciculi become promptly perceptible.

According to MM. Mannkopf[2] and Engelken,[3] these lesions are chiefly remarkable on account of the proliferation of the nuclei of the sarcolemma. They bear, in fact, the stamp of the irritative process. Here, also, fatty degeneration of the primitive fasciculi is an exceptional incident. As to the nerves which supply the affected muscles, they were found by Herr Mannkopf, after repeated examinations, to be sometimes healthy, sometimes affected by comparatively slight alterations nowise proportionate in intensity to the severity of the muscular lesions.[4]

Spinal apoplexy (*hæmatomyelia*) should be mentioned in the second place. This is an affection which, considered from the standpoints of pathogeny and pathological anatomy, differs essentially from common intra-cerebral hemorrhage; for, in hæmatomyelia, the effusion usually takes place in the midst of tissues which have already suffered modification from inflammatory action. The blood is chiefly effused in the gray matter, which it often invades throughout the major part of its length. When this happens, diminution or even abolition of electrical contractility, supervening early in the muscles of the paralyzed members, is a symptom which seems constant. It was observed fourteen days after the development of the first accident, by Levier;[5] on the very day of the attack, by Colin (?); on the ninth day, in a case recorded by Duriau.[6] Spinal apoplexy is an affection which, in general, is rapidly fatal; it has as yet furnished no opportunity of observing the histological lesion of the primitive fasciculi and the atrophy of the muscular masses, which would doubtless not fail to follow if life were prolonged.

It is probable, gentlemen, that *fractures and luxations of the verte-*

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[1] Mannkopf, 'Amtlicher Bericht über die Versammlung Deutscher Naturforscher und Aerzte zu Hannover,' p. 251. Hannover, 1866.

[2] Mannkopf, *loc. cit.*

[3] Engelken, 'Beitrag zur Patholog. der acuten Myelitis.' Zurich, 1867.

[4] *Vide supra*, p. 36.

[5] Levier 'Beitrag zur Pathologie der Rückenmarksapoplexie.' Inaugural-dissertation. Bern, 1864.

[6] Duriau, 'Union Médicale,' t. i. 1859, p. 308.

bral column, by producing an irritation of the cord which from partial tends to become general, may have the effect of determining, as Dr. Duchenne (de Boulogne[1]) has remarked, a prompt diminution of electrical contractility in the muscles of the paralyzed member.

2d. The affections which compose the second category are the product of more delicate lesions. These are, in fact, limited, in a seemingly systematic manner, to the gray matter of the anterior cornua, the entire extent of which they rarely invade; we see them often very exactly localized in the circumscribed oval space occupied by a group or cluster of motor cells (Fig. 2).

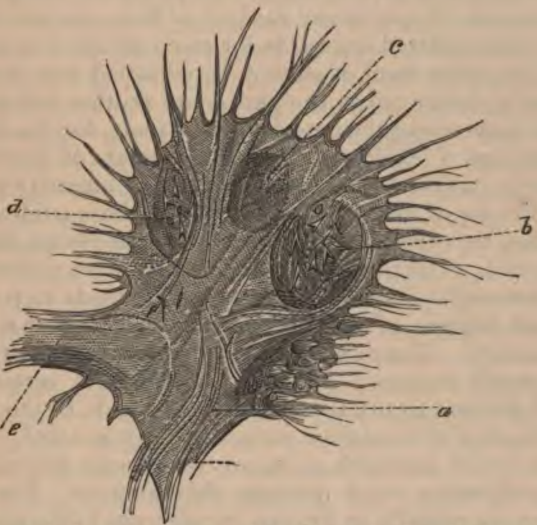

FIG. 2.—Fragment of a transverse section of the spinal cord taken from the lumbar region, in a case of infantile spinal paralysis occupying the right inferior extremity. The right anterior cornu of gray matter is represented. The lesions affect exclusively the antero-external group of nerve-cells: *a*, *cervix cornu posterioris*; *b*, postero-external group of nerve-cells; *c*, antero-external group. The cells of the latter group have completely disappeared, whereas those of groups *b* and *d* are perfectly distinct; *d*, internal group; *e*, the commissure.

The neuroglia, in the affected parts, becomes usually opaque, denser, strewn with numerous myelocytes and consequently exhibits the signs of inflammatory action. At the same time, the nerve-cells present different degrees and different modes of atrophic degeneration. But what are the elements first affected? Everything leads us to believe these to be the nerve cells. It would, indeed, be difficult to comprehend how the alteration could show itself strictly localized in the vicinity of the cells if its starting point were in the neuroglia. There are cases besides, in which the atrophy of a certain number, or even of a whole group, of nerve-cells is the only alteration which can be discerned, on histological examination. The connective web preserves its transparency, in those points, and all the characters of normal structure, with but few exceptions. There are other not less significant cases, also, where lesions of the neuroglia appear much more developed about the central parts of a cluster of nerve-cells, than in the peripheral portion, and also much more manifest in the immediate neighbourhood of the cells themselves, than in the intervals between them, so that they appear like so many centres or foci, whence the inflammatory process radiated outwards for some distance in every direction. On the other hand, it cannot be admitted that the irritation was originally developed in the peripheral portion and ascended to the central parts along the anterior nerve-roots, for the latter generally present, on a level with the altered points of the cords, only comparatively trifling lesions and not at all of intensity proportionate to the lesions of the gray matter. It appears evident, from all that precedes, that the motor nerve-cells are, in reality, the primary seat of the evil. Usually, it is true, the irritative process next invades the neuroglia, its second stage; and extends step by step to the different regions of the anterior cornua; but that is by no means necessary. Hence we must, *a fortiori*, regard as a consecutive and purely accessory fact, the extension, in certain cases, of the morbid process to the antero-lateral fasciculi, in the immediate neighbourhood of the anterior cornua of gray matter.[1]

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[1] Duchenne (de Boulogne), Observation, p. 246, *loc. cit.*, fracture of the vertebral column, about the middle of the dorsal region. The spinal cord softened for several inches, in the dorso-lumbar region. Enfeeblement of the electrical contractility from the sixth day after the accident.

Infantile spinal paralysis is, up to the present, the most perfect type of the affections which compose this category. The numerous researches made recently in France, in reference to the spinal lesions on which they depend, concur to indicate, as an essential fact, the profound alteration of a large number of motor-cells, in those regions of the cord whence the nerves emanate which supply the paralyzed muscles.[2] In the vicinity of the atrophied cells, the connective network almost always offers manifest traces of an inflammatory process. Judging from the general aspect of the phenomena, we are induced to admit, as a highly probable hypothesis, that, in infantile spinal paralysis, a superacute irritative action suddenly seizes on a large number of nerve-cells and makes them promptly lose their motor functions. Some cells, which have been but slightly attacked, will recover their functions some day, and this phase corresponds to the amelioration of symptoms which always supervenes at a certain period of the disease. Others, however, have been more severely involved, and the irritation of which they were the seat is transmitted along the nerves to the paralyzed muscles which, in consequence, suffer trophic lesions of a more or less serious character.[1] However it be, it is known that diminution or even loss of faradaic contractility may be observed, in certain muscles, barely five or six days after the abrupt invasion of the first symptoms. The emaciation of the muscular mass makes rapid progress, besides, and soon becomes evident. The alterations which, on histological examination, are found in the affected muscles are these: firstly, simple atrophy of the primitive fasciculi with the transverse striæ preserved; and, secondly, the marks of a more or less active proliferation of sarcolemma-nuclei on some isolated fasciculi. The accumulation of fat, sometimes seen in old cases, seems to be a purely adventitious phenomenon.[2]

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[1] The views set forth above relative to the rôle of the so-called motor nerve-cells in the pathogeny of progressive muscular atrophy, infantile paralysis, acute central myelitis, and of all the amyotrophies arising from spinal causes, in general, were stated in a lecture which I delivered at La Salpêtrière in June, 1868. *Cf.* Hayem. 'Archives de Physiologie,' 1869, p. 263. Charcot et Joffroy, *id.*, p. 756. Duchenne (de Boulogne) and Joffroy, *id.*, 1870. These views have been utilized in the recent work of Dr. Hammond, 'A Treatise on Diseases of the Nervous System,' Sect. iv. 'Diseases of Nerve-cells,' p. 683, New York, 1871.

[2] On atrophy of the motor nerve-cells in infantile paralysis, consult Prevost, in 'Comptes Rendus de la Société de Biologie,' 1866, p. 215. Charcot et Joffroy, "Cas de Paralysie Infantile Spinale, avec lesions des cornes antérieures de la substance grise de la moelle épinière," in 'Archives de Physiologie,' p. 135, 1870, pls. v et vi. Parrot et Joffroy, *id.*, p. 309. Vulpian, *id.*, p. 316. H. Roger et Damaschino, "Recherches Anatomiques sur la Paralysie Spinale de l'Enfance," in 'Gazette Médicale,' Nos. 41, 43, et suiv., 1870 (fig. 2).

Progressive muscular atrophy offers for our study the irritative atrophy of the motor cells in its chronic form.[3] Here we are not concerned with a superacute irritative process suddenly invading the nerve-cells, and laying hold on a large number of them. These are now affected successively, one by one, in a progressive manner; a considerable number of them are spared, even in the most seriously compromised regions, until nearly the last period of the disease. The development of the muscular lesions corresponds to this mode of evolution of the spinal lesions. Thus, it is rare that the trophic disorders affect simultaneously all the primitive fasciculi of a muscle; hence it follows that the latter may respond, in a more or less imperfect manner to the command of the Will, and still enter into contraction under the influence of electrical excitations, even when its volume has been very markedly diminished.[4]

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[1] Charcot et Joffroy, *loc. cit.*

[2] *Ibid.*, *loc. cit.*; Vulpian, *loc. cit.*

[3] On atrophy of the motor-cells in progressive muscular atrophy, see Luys, 'Sociétié de Biologie,' 1860. Duménil (de Rouen), "Atrophie musculaire graisseuse progressive, historie critique," Rouen, 1867. "Nouveaux faits relatifs à la pathogenie de l'atrophie musculaire progressive," in 'Gazette hebdom.,' Paris, 1867. Lockhart Clarke, "On a Case of Muscular Atrophy," 'British and Foreign Medico-Chirurigical Review,' July, 1872. "A Case of Muscular Atrophy," Beale's 'Archives,' t. iv, 1867. "On a case of Muscular Atrophy," in 'Medico-Chir. Transactions,' t. iv, 1867. O. Schüppel, "Ueber Hydromilus," in 'Archiv der Heilkunde,' Leipzig, 1865, p. 289. Hayem, in 'Archives de Physiologie,' 1869, p. 263, pl. 7. Charcot et Joffroy, in 'Archives de Physiologie,' 1869, p. 355.

[4] Charcot, "Leçons faites à la Salpêtrière in 1870." See also Hallopeau, in 'Archives de Médecine,' Septembre, 1871, pp. 277, 305.

There exist, indeed, at least two very distinct forms of progressive amyotrophy correlated to an irritative lesion of motor cells. One of them, which is *protopathic*, arises exclusively from the lesion in question, and this form, primarily developed because of an original or acquired predisposition, tends, as of necessity, to become generalized. In the other form, to which we called your attention a moment ago, the nerve-cell is only secondarily affected, consecutively on a lesion of the white fasciculi, for instance, and as it were accidentally. Progressive amyotrophy, in the latter case, may perhaps be called *symptomatic*, it has less tendency to become generalized, and its prognosis is certainly less gloomy.[1]

As regards *adult spinal paralysis*, and *general spinal paralysis* (Duchenne de Boulogne), pathological anatomy has not yet given any definite decision. But to judge from the symptoms, it is at least very probable that these affections also depend on a lesion of the motor nerve-cells of the anterior cornua. Adult spinal paralysis resembles that of childhood by the almost sudden invasion of motor paralysis, by the tendency which it shows to retrogade at a given moment, by the diminution or abolition of faradaic contractility showing itself precociously in a certain number of paralyzed muscles, and, finally, by the rapid atrophy which these same muscles constantly exhibit to a more or less marked extent. A slower evolution, often occurring in a subacute or chronic manner, a tendency to become generalized, especially evident in the first stages, frequent pauses, followed by invasion of hitherto untouched parts, distinguish, on the contrary, general spinal paralysis, and make it resemble progressive muscular atrophy, with which it is sometimes, very erroneously, confounded in clinical practice. The former, however, is clearly separated from the latter by the following characters: the muscles of an entire extremity or of portion of a limb, are struck, *en masse*, in an almost uniform manner, with paralysis or atrophy; they present, at a period but little remote from the commencement of the disease, very marked modifications of electrical contractility; usually, in conclusion, a period of recovery supervenes, during which the atrophied muscles regain, at least partially, their volume and their functions.[2]

Muscular lesions consecutive on affections of the bulbus rachidicus.—This is a subject which has been, as yet, but little explored. However some facts, which have accumulated until they now form a respectable number, gleaned from the history of labio-glosso-laryngeal paralysis and disseminated sclerosis (*en plaques*) tend to establish that, in the case of the bulbus as well as in that of the spinal cord, irritative lesions of the white fasciculi have no direct influence on the nutrition of muscles. Those, on the contrary, which affect the motor cell clusters distributed over the floor of the fourth ventricle, or the fasciculi of nerve-tubes emanating from these aggregations may, as I have demonstrated, determine a more or less marked atrophy of the muscular fibres of the tongue, pharynx, larynx, and orbicularis oris.[1]

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[1] On the two forms of progressive amyotrophy of spinal origin, see Charcot et Joffroy in 'Archives de Physiologie,' 1869, pp. 756, 757. Duchenne (de Boulogne) et Joffroy in 'Archives de Physiologie,' 1870, p. 499.

[2] Duchenne (de Boulogne), 'De l'electrisation localisée,' 3d edition.

The summarized account which has been laid before you will suffice, I hope, to place in prominence the remarkable rôle which, according to the most recent researches, lesions of the anterior nerve-cells play in the production of trophic muscular disorders consecutive on alterations of the spinal cord. This rôle does not seem doubtful in the pathogeny of infantile paralysis and of the different forms of amyotrophy of spinal origin. Its influence is, certainly, less distinctly demonstrated, though still highly probable, as regards hæmatomyelia, acute central myelitis, and, in a word, all the irritative affections of the spinal cord in which the gray axis is found to be involved. On the other hand, the absence of all participation on the part of the white fasciculi, and of the posterior cornua, in the development of the muscular affections in question is a fact which henceforth rests on abundant evidence.

This acknowledged, gentlemen, we have cause to inquire why lesion of the motor nerve-cells induces that of the muscular fibres, whilst even the gravest irritative alterations of the white fasciculi have no direct influence on the nutrition of the muscles.

With respect to the first point, one cannot fail to imagine more or less plausible hypotheses which, however, are evidently premature. The teachings of experimental physiology cannot be called to our assistance here; its methods of procedure, inferior to those of disease in that respect, are not sufficiently delicate to allow the nerve-cells to be attacked in an isolated manner. We must, therefore, confine ourselves, at present, to registering the facts as they are offered us in clinical practice, illustrated by pathological anatomy, and to point out that the motor nerve-cells, comparable in that respect to the peripheral nerves, possess the power, when they have become the seat of irritation of modifying, by remote action, the vitality and structure of the muscles.

As regards the second point, if what we have said concerning the effects of nerve-irritation be referred to, it may seem contradictory, at first sight, that the nutrition of the muscles should not be affected when the white fasciculi of the cord are occupied by inflammation. To show that the contradiction is only apparent it will suffice, however, to remind you that, in spite of the analogy of com-

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[1] Compare Charcot, "Note sur un cas de paralysie glosso-layngée, suivi d'autopsie," in 'Archives de Physiologie,' 1869, pp. 356, 636, pl. xiii. Obs. de Catherine Aubel. Duchenne (de Boulogne) et Joffroy, "De l'atrophie aïgue et chronique des cellules nerveuses de la moelle et du bulbe rachidien," 'Archives de Physiologie,' 1870, p. 499.

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position, the white columns are not at all comparable to the nerves. Experiments in fact, reveal, in the latter, properties which are not to be found in the former, and *vice versâ*. Anatomy also shows that the nerve-tubes which constitute the nerves are but to a very small extent the direct continuation of those which, by their union, form the white substance of the cord. These fasciculi appear to be almost entirely composed of fibres which, arising either in the encephalon or in the cord itself, establish, after the manner of commissures, communications between the spinal cord and the brain, or between different points of the gray spinal axis. It was to be anticipated, from this, that, in many respects, the white fasciculi of the cord would, under the influence of irritative lesions, behave differently from the peripheral nerves.

When I formed the idea of laying before you, gentlemen, the principal facts relating to the nutritive disorders which make their appearance consecutively on affections of the nervous system, I hoped that my task might be brought fairly to an end, in the course of two lectures. But, according as I advance in this exposition, the importance and extent of the question display themselves in all their distinctness. Notwithstanding the details which I have already given, I am far from having exhausted the subject, and I dare to hope that you will not have cause to regret the time that yet remains to be dedicated to its study.

LECTURE III.

DISORDERS OF NUTRITION CONSECUTIVE ON LESIONS OF TH SPINAL CORD AND BRAIN.

SUMMARY.—Cutaneous affections in sclerosis of the posterior columns: papular or lichenoid eruptions, urticaria, zona, pustular eruptions; their relations with the fulgurant pains; the former appear to arise from the same organic cause as the latter.

Eschars of rapid development (acute bed-sores) in diseases of the brain and spinal cord. Mode of evolution of this skin-affection: erythema, bullæ, mortification of the derma, accidents consecutive on the formation of eschars: *a*, putrid infection, purulent infection, gangrenous emboli; *b*, simple purulent ascending meningitis, ichorous ascending meningitis. Acute bed-sore in apoplexy symptomatic of circumscribed cerebral lesions. It appears principally in the gluteal region of paralyzed extremities; its importance in prognosis. Acute bed-sore in diseases of the spinal cord; it generally occupies the sacral region.

Arthropathies depending on a lesion of the brain or spinal cord. *A.* Acute or subacute forms; they appear in cases of traumatic lesion of the spinal cord; in myelitis occasioned by compression (tumours, Pott's disease), in primary myelitis, in recent hemiplegia, connected with cerebral softening. These arthropathies occupy the joints of paralyzed limbs. *B.* Chronic forms; they seem to depend, like amyotrophies of spinal origin, on a lesion of the anterior cornua of the gray axis; observed in posterior sclerosis (locomotor ataxia) and in certain cases of progressive muscular atrophy.

GENTLEMEN: In treating of the nutritive disorders determined by lesions of the peripheral nerves, I gave you to expect that these consecutive affections would, for the most part, be represented in cases of lesions of the spinal axis. It is true, we shall not always find here a servile imitation; indeed, as a general rule, the trophic disorders of cerebral or spinal origin, as we shall often have occasion to note, bear with them the distinctive stamp of their cause. But there are circumstances in which the resemblance between affections of central origin and those which depend on a lesion of the peripheral nerves is so striking that discrimination may be a most difficult task. We will cite, as examples of this class, certain cutaneous eruptions which sometimes supervene in the course of ataxia.

I.

The *cutaneous affections*, to which we have just alluded, may be classified as follows: *a*, *papular* or *lichenoid eruptions*; *b*, *urticaria*; *c*, *zona*; *d*, *pustular eruptions*, analogous to ecthyma.

The following, in a few words, are the results of my observations on this subject. It is not rare to see the skin of the legs and thighs become temporarily covered with a more or less confluent papular or lichenoid eruption, consequent on paroxysms of the fulgurant or shooting pains, characteristic of locomotor ataxy. In the case of a woman, at present under treatment at La Salpêtrière, enormous patches of urticaria are produced, at every paroxysm, over the parts where the keenest pains are felt. In another case, the skin of the right gluteal region becomes covered with an herpetic eruption, limited however to the course of the nervous filaments which convey the pain. Finally, a third patient presented, under analogous circumstances, still more remarkable phenomena. This woman, aged sixty-one years, was received into the hospital on account of blindness (sclerous atrophy of the optic nerves) about eight years ago; she is now suffering from well-marked locomotor ataxia. In her case, the evolution of the disease has been very rapid, for the first paroxysms of shooting pains date from the month of March, 1865, and in July, 1866, the incoördination was so far advanced as to render walking difficult. One of these fits, which happened in June, 1867, was of exceptional intensity. The pains which were really horrible, seemed fixed, during several days, along the course of the cutaneous branches of the right lesser ischiatic nerve, and of that supplying the levator ani.[1]

During this time, the corresponding parts of the skin became covered with a great number of pustules, analogous to ecthyma, some of which proved the starting points of deep ulcerations. Besides this, a rounded eschar of about two inches in diameter, which involved the derm nearly throughout its whole thickness, developed in the right sacral region, a few inches from the median line immediately under the extremity of the coccyx. The sore persisting after the elimination of the sphacelated parts, cicatrization was not complete until two months had elapsed. In another paroxysm, the flashing pains followed the direction of the vertical portion of the left internal saphenous nerve, and a pustular eruption was soon thrown out on the skin of the regions to which this nerve is distributed.

There is one character common to all these eruptions, and it is of a kind to show that we have not here to deal with common disorders,—they all make their appearance concurrently with certain intense and persistent exacerbations of the specific pains, which are in some sort pathognomonic of fasciculated sclerosis of the posterior columns, which it is customary to call fulgurant or flashing pains.

Let me add, as another character, that the eruptions in question habitually show themselves along the course of the nerves invaded by the fulgurating pain.

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[1] The nerve called "releveur de l'anus" by French anatomists, is a branch of the fourth anterior sacral nerve, although the muscle, bearing the same name, receives twigs from the superficial perineal nerve (S.).

From what precedes you will observe that the existence of those cutaneous eruptions seems closely connected with that of the fulgurant pains: hence it becomes at least very probable that one and the same organic cause presides over the development of both the former and the latter.

What, then, is the reason of the presence of fulgurant pains among the symptoms of sclerosis of the posterior columns? I do not desire to enter to-day upon a long discussion of this question which will meet us again; it will suffice, at present, to tell you that, in all probability, these pains depend upon the irritation set up, during their intra-spinal course, in those of the nerve-tubes emanating from the posterior roots which, under the name of internal radicular fasciculi (*internal fibrous masses of the posterior roots* in the nomenclature of Kölliker),[1] pass, for a certain extent, through the area of the posterior columns before penetrating the posterior cornua of the gray matter.

It appears but little possible to connect the fulgurant pains with any one of the following lesions: 1st, atrophy of posterior roots, before entering the cord; 2d, posterior spinal meningitis; 3d, sclerosis of the posterior cornua of the gray matter; 4th, irritative lesions of the spinal ganglia or of the peripheral nerves,—for these pains have been met with in a certain number of ataxic cases in which post-mortem examinations have demonstrated the absence of all lesions of the kinds enumerated.

In support of this proposition allow me, gentlemen, to recall the results of the autopsy which Dr. Bouchard and I made, in the case of a woman who died in this hospital, during the first period of progressive locomotor ataxia.[2] This patient had experienced the special paroxysmal pains, in a high degree, lasting for some fifteen years, until the epoch of her death caused by an adventitious disease. No sign of motor incoördination had ever shown itself. The patient walked with ease, without throwing forward the leg, or stamping with the heel, nor did closing the eyes affect her certainty of movement. On post-mortem examination, we saw that the posterior roots had preserved their normal characters, and beyond some equivocal traces of meningitis, the only perceptible lesions met with occupied the posterior columns, and consisted of a multiplication of neuroglia-nuclei with thickening of the meshes of reticulum, but without concomitant alteration of the nerve-tubes. To complete the demonstration, I could cite many cases of the same kind where the fulgurant pains had been likewise very intense, and where, on a post-mortem examination, I was unable to discover the existence of any alteration whatever, either in the posterior gray cornua, or in the peripheral nerves, or in the spinal meninges.

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[1] Kölliker, 'Histologie Humaine,' P. i, pp. 345, 346.

[2] "Douleurs fulgurantes de l'ataxie, sans incoördination des mouvements, sclérose commençante des cordons postérieurs de la moëlle épinière," in 'Comptes Rendus des Séances et Mémoires de la Société de Biologie,' 1866.

From this it would appear necessary that we should seek, in the irritative alteration of the posterior columns of the spinal cord, the starting point of the fulgurant pains of ataxic patients. But it seems scarcely probable that all parts of these fasciculi ought to be indiscriminately arraigned on this count: everything, on the contrary, induces us to believe that the sensitive fibres, issuing from the posterior roots, which compose a portion of the *internal radicular fasciculi* should alone be incriminated. These fibres would participate, from time to time, periodically, in the irritation whose permanent seat is in the columns themselves; and thus would be produced those paroxysms of shooting or flashing pains which, in accordance with a well-known physiological law are referred to the periphery, although in reality due to a central cause.

How are we to understand the appearance of the cutaneous eruptions sometimes observed in ataxic patients, at the very time of the occurrence of fulgurant paroxysms of abnormal intensity? It is certain that the nerve fibres which form the internal radicular fasciculi are not all sensitive; there are, for instance, at least some amongst them which assist in the accomplishment of reflex actions; there are others also, no doubt, at least it is what these cutaneous eruptions tend to demonstrate, which belong to the system of centrifugal nerves and which possess a more or less direct influence over the exercise of the nutritive functions of the skin. The irritation of the latter class of fibres, an irritation more difficult to set up than that of the sensitive fibres, should be invoked to explain, in the cases I have above alluded to, the production of papular affections at one time, and at another, of vesicular, pustular, or gangrenous disorder.

Are the posterior fasciculi the only departments of the spinal cord, the irritation of which is capable of determining such affections? This is a question which must remain unanswered for the present. All that can be said is that such eruptions have not yet been observed, except where there was some complication, in cases of irritative lesions confined to the antero-lateral columns, or to the anterior cornua of the gray matter; and as to the part which the posterior cornua may play, in this respect, we are in the most complete ignorance upon that subject.

On the other hand, some facts have been collected which tend to establish that zona is sometimes developed under the direct influence of partial lesions of the encephalon. Thus, in the case of an aged woman attacked with hemiplegia, whose history has been recorded by Dr. Duncan, an eruption of zona appeared on the thigh of the paralyzed side; motor paralysis had supervened almost simultaneously with the eruption, and both passed away nearly at the same time.[1]

In the case of a child, recorded by Dr. Payne, the zona, which marked out the course of superficial branches of the anterior crural nerve, showed itself three days after the development of a hemiplegia occupying the same side of the body as the eruption.[2] These cases, which can be multiplied, are undoubtedly very interesting; unfortunately, they have been related in a very summary manner only, and caution is needed, I think, in drawing conclusions from them, which may prove premature. I can, in fact, cite a case in many respects analogous to the preceding, which I recently observed at La Salpêtrière, and where the cause of the zona was most probably the irritation of a peripheral nerve. Here, again, the seat of the vesicular eruption was in the inferior extremity of the paralyzed side, where it followed the distribution of the superficial twigs of the cutaneous perineal nerve. It showed itself also, simultaneously with the hemiplegia which, making an abrupt appearance, was correlated to the formation in one of the cerebral hemispheres of a focus of ramollissement, itself being determined by the embolic obliteration of a posterior cerebral artery. As to the zona, it was produced, I believe, after the following mechanism, a spinal arterial branch,[3] arising, no doubt, from one of the lateral sacral arteries was, on a post-mortem examination, found to be obstructed by a blood-clot, and to form a comparatively voluminous cord, adhering to one of the posterior spinal roots of the cauda equina. It is probable that, on its passage through the sacral foramen, this arteriole, exceedingly distended by the thrombus, had compressed either the spinal ganglion, or an initial branch of the ischiatic nerve, so as to set up irritation in it. A vegetating ulceration, which was noticed on one of the sigmoid valves of the aorta, appears to have been the starting point of all the accidents which we have just described.[4]

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[1] 'Journal of Cutaneous Medicine,' &c., 69. Erasmus Wilson, October, 1868.

[2] 'British Medical Journal,' August, 1871.

[3] One of the *rami medullæ spinalis*, see N. Rudinger, "Arterienverzweigung, in dem Wirbelcanel, &c.," in 'Verbreitung des Sympathicus,' p. 2, München, 1863.

[4] The following are the principal details of this case which presents a fine example of ulcerous endocarditis, with multiple emboli and a typhoid condition.

The patient Lacq, ... aged 22 years, a soldier, was admitted on the 28th December, 1870, to the Salpêtrière ambulance (fever ward). He had been suffering, it seems, from an intense fever for two or three days. On the day of admission the following symptoms were noted: severe cephalalgia, pains in the loins, diarrhœa. The patient cannot swallow the smallest quantity of liquid without being taken with nausea and vomiting. Skin hot, pulse very frequent. It was regarded as a case of typhoid fever. Noisy delirium during the night. Next day, 29th December, was noticed the existence of an almost complete hemiplegia of the left side. The paralyzed members were not rigid; incomplete facial paralysis, on the left side, likewise existed. The eyes are constantly directed to the right side, and there is nystagmus. Pulse 120; rectal temperature 40.5° Cent. On the breast, forearms, and thighs, the skin shows a great number of little ecchy-

You will observe that, in this case, the coexistence of the hemiplegia and of the vesicular eruption resulted to a certain extent from a fortuitous coincidence. However it be, in default of zona, there are other trophic disorders of the skin, the existence of which may sometimes be attributed to the influence of an encephalic lesion. This is a fact which, I hope at least, will soon be placed beyond doubt.

II.

Eschars of rapid development. Decubitus acutus: acute bed-sore.[1]—I hasten to leave the questions of eruptions occurring in locomotor ataxia, which, on the whole, have but a secondary importance, in order to draw your attention, in a very special manner, to another affection of the skin which holds a most important position in the clinical history of a considerable number of the diseases of the brain and spinal cord.

The cutaneous affection, which I am about to discuss, shows

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moses, somewhat resembling flea-bites,—frequent respirations, sibilant râles.—Tympanites. On the antero-external surface of the left paralyzed leg, there exists an eruption of zona which answers exactly to the distribution of the superficial twigs of the cutaneous perineal branch of the musculo-cutaneous nerve. The first group of vesicles is seen above and below the patella; a larger group is disposed in a vertical straight line which descends to the middle third of the leg; the third group occupies the neck of the foot before and inside of the external malleolus. The eruption is tolerably developed. It is remarked that some traces of it existed the day before, that is to say, previous to the hemiplegia. On the 30th, the eruption is in full vigour. The patient succumbs at 4 o'clock in the afternoon.

Autopsy.—One of the sigmoid valves of the aorta is ulcerated and covered with vegetations, fibrinous, soft, and reddish in appearance. The mesenteric glands are somewhat red and swollen, but there exists no trace of dothienenterical eruptions or ulcerations in the small or large intestines. Numerous ecchymoses are observed on the visceral and parietal pleuræ, in the pericardium, and in the peritoneum. The spleen and kidneys present infarcti in different stages of development. Right cerebral hemisphere; on many points of the occipital lobe the pia mater, which is much injected, presents large patches of sanguine suffusion. The lobe itself is softened throughout nearly its whole extent; the cerebral matter there assumes a grayish colour, and at one point in the midst of the softened parts we note an effusion of blood, as big as an almond. The posterior cerebral artery of the same side is completely obliterated by a thrombus. The spinal cord, prepared with chromic acid, and examined in thin sections, presents no perceptible alterations. At the cauda equina, on the left side, we found adhering to one of the posterior spinal roots which give origin to the sacral plexus, an arteriole (spinal branch, arising from the lateral sacral artery) distended by a blood-clot. The obliterated artery, equal in size to a crow-quill, may be followed from the point where the root has been cut not far from the corresponding sacral foramen, to the spinal cord; upon this it can still further be followed the whole length of the lumbar enlargement, where it ascends along the posterior median fissure, contrary to the usual arrangement of the posterior spinal arterial plexus.

[1] *Decubitus* (when qualified by the adjectives *acutus, chronicus, ominosus*) signifies, not the position of the patient in bed, but the bed-sores supposed to result from such position. This term, though etymologically objectionable, is generally employed in foreign hospitals. As its adoption in this translation might confuse, and appears to be unnecessary, the term "bed-sore" has been substituted (S.).

itself at first under the form of an erythematous patch, on which vesiculæ and bullæ are rapidly developed; it terminates very often in mortification of the skin and subjacent tissues.

Usually it occupies the sacro-gluteal regions; but it may also appear almost indifferently on all parts of the trunk or members subjected, in the decubitus, to a somewhat continuous pressure. Even a very slight and very short pressure suffices to make it appear in certain cases. Finally, there are other cases still, though these indeed are very exceptional, in which it seems to be produced without the intervention of the least pressure or of any other occasional cause of the same kind.[1]

This is a very different affection from all those various eruptions which are so commonly remarked over the sacrum in patients condemned by different disorders to long maintain a recumbent position in bed. These eruptions which are sometimes erythematous and lichenoid, sometimes pustular and ulcerous, sometimes papular, having a deceptive resemblance to syphilitic sores (*plaques muqueuses*), are generally occasioned by repeated and prolonged contact with irritating substances, such as urine or fecal matters. They, as well as acute bed-sore, may become the starting points of genuine eschars; but the acute bed-sore is distinguished from the former by important characters, namely: firstly, by appearing shortly after the commencement of the primary disease, or following on a sudden exacerbation; and, secondly, by a very rapid evolution.

On account of the peculiar interest belonging to it, the affection, in question, certainly deserves to be designated by an appellation proper to itself. One of the few authors who have made it a special study, Herr Samuel, has proposed to characterize it by the name of *decubitus acutus* or eschar of rapid formation. He desires thus to distinguish it from *decubitus chronicus*, that is, from the dermal necrosis which appears long after the invasion of the disease which occasions its existence. We propose to accept this appellation, whilst reminding you, however, that the mortification of the skin is not everything in *decubitus acutus*.[2] It answers, on the whole, to the most advanced phases of the morbid process. It may happen, indeed, that the vesiculæ or bullæ will dry up and disappear without that portion of the derm, on which they were seated, presenting the least trace of necrosis. This is principally observed when they are produced on parts where the pressure has only been of short duration, of little intensity, and, so to speak, accidental, as over the ankles, on the inner surface of the knees, the legs, or the thighs. Now, it behooves you to be able to recognize the significance of these vesiculæ and bullæ, from their first appearance on the scene; for, even at that period, they enable us under certain circumstances to formulate a prognosis, with certainty.

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[1] Brown-Séquard, 'Lectures on the Central Nervous System,' Philadelphia, 1868, p. 248. Couyba, 'Des Troubles Trophiques,' &c., Thèse de Paris, 1871, p. 43.

[2] See note 1, p. 56.

The opportunity has been given me, many times, of following as it were day by day, hour by hour, the evolution of the *acute bed-sore*, in cases of apoplexy consecutive on hemorrhage, or on softening of the brain which we so often meet with in this hospital.[1]

I can refer to the observations I made in regard to this, in the general description which follows, for I have been able to establish, from another stand-point, that the acute bed-sore connected with brain-diseases does not essentially differ from that which arises under the influence of spinal lesions.

Some days or even some hours only after the manifestation of the cerebral or spinal affection, or again, following on a sudden exacerbation of these affections, there appear on certain points of the skin one or many erythematous patches, variable in extent and irregular in shape.[2] The skin there has a rosy hue, sometimes it is dark red, and even violet, but the colour disappears momentarily on pressure with the finger. Under somewhat rare conditions, which hitherto I have met with almost entirely in cases of spinal lesions, there appears besides, involving the derm and subjacent tissues, an *apparently phlegmonous tumefaction*, which may be accompanied sometimes by acute pain, if the region has not been previously smitten with anæsthesia.

On the morrow, or after-morrow, vesiculæ or bullæ make their appearance towards the central part of the erythematous patch; they contain a liquid, sometimes colourless and perfectly transparent, sometimes more or less opaque, reddish, or brown-coloured.

Matters may remain so, as we have already mentioned, and then the vesicles and blebs soon wither, dry up, and disappear. At other times, however, the elevated epidermis becomes torn, drops off in pieces, and lays bare a bright red surface strewn with bluish and violet points or patches, corresponding with a sanguine infiltration of the derm. In such cases the subcutaneous connective tissue, and sometimes even the subjacent muscles are themselves already invaded by sanguine infiltration. This fact I have repeatedly verified by post-mortem examination.

The violet patches extend rapidly in width and their edges soon run together and unite. A short time after, there supervenes in the affected part, a mortification of the derm which, at first superficial, soon grows profound. From that time, the eschar is constituted. Later on comes the development of the work of reaction and elimination, followed, in favourable cases, by a period of reparation which is too often impeded in its course. It is unnecessary for me, I think, to expatiate on this point.

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[1] Charcot, "Note sur la formation rapide d'une eschare à la fesse du côte paralysé dans l'hémiplégie récente de cause cérébrale," 'Archives de Physiol. normale et pathol.,' t. i, 1868, p. 308.

[2] I have ascertained, anatomically, that in such cases the derm is infiltrated with leucocytes, as happens in erysipelas.

I have been occupying your attention with minute details, but I trust I shall induce you to acknowledge that they have their own peculiar interest. R. Bright thought them sufficiently worthy of notice and novel enough to believe he should insist upon them in his "Reports of Medical Cases," and should get wax models made of the bullæ of *acute bed sore* observed in a case of traumatic paraplegia.[1] These models still figure, no doubt, in the museum of Guy's Hospital.

Since then, as far as I know, this subject has but slightly arrested the attention of observers, with a few rare exceptions.[2] It would be unjust, however, not to acknowledge that, in cases of typhus and typhoid fevers, a cutaneous affection, which offers the closest analogies with this disorder and which, perhaps, partly depends on analogous conditions, has been minutely described in France by Piorry,[3] and in Germany, by Pfeüfer.[4]

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[1] It will not be deemed inappropriate to quote here the remarks which R. Bright has appended to his cases of affections of the spinal cord, with rapid formation of bullæ and eschars, which he has consigned to his "Reports of Medical Cases," (t. ii, 'Diseases of the Brain and Nervous System,' London, 1831). First comes a case where softening of the spinal cord supervened, without any known external cause, in a young woman aged 21; the lesion occupied the lumbar enlargement immediately above the cauda equina. The case suggested the following reflections:—

"Another curious circumstance connected with paralysis of the lower extremities is illustrated by this case: the tendency which is observed in such affections to the formation of vesications or bullæ, which frequently make their appearance in a night, on some part, as the knee, the ankle, or the instep, where accidental pressure or irritation has taken place; they contain a limpid fluid which after a few days becomes opaque. It has sometimes struck me that this connection between interrupted nervous action and the formation of bullæ, might hereafter be found to throw light on that most singular disease herpes zoster which, from the peculiar pain with which it is accompanied, as well as from its strict confinement to one side of the body, seems to be connected with some peculiar condition, perhaps the distension of the sentient nerves." (p. 383.)

Three other cases relating to traumatic lesions of the spinal cord (caused by a fall from a height, the passage of a wagon, &c.) are commented on as follows:—

"The two most remarkable points to be incidentally noticed in the foregoing cases are, first, the diseased state of the bladder, resulting from its diminished power to resist injury, and from the changes taking place in the condition of the urine, detained in its most depending part, which becomes one of the most frequent causes of fatal irritation in paraplegia;—and secondly, we observe the occurrence of bullæ on the paralyzed limbs, to which circumstance I have already alluded in some remarks made at p. 383; the general inability to resist injury is likewise marked by extensive sloughing of all the paralyzed parts on which pressure is made." (p. 423.)

[2] After R. Bright, we must specially refer to Sir Benjamin Brodie ("Injuries of the Spinal Cord," 'Med.-Chir. Transactions,' t. xx, 1837), and Brown-Séquard (*loc cit.*).

[3] A. Touzé, "Des dermopathies et des dermonécroses sacro-coccygiennes," Thèses de Paris, 1853.

[4] Kerchensteiner's "Bericht," in 'Henle und Pfeüfer's Zeitschrift für rationelle Medicin,' Bd. v. See also Wunderlich, 'Pathologie,' t. ii, p. 285.

Let us return, gentlemen, to the *bed-sore* provoked by diseases of the nervous centres. You know too well the accidents which eschars, from whatever cause arising, are capable of engendering for me to indulge in a detailed description. Allow me, however, to sketch out in a few words the principal amongst them, for you must expect to see them often figuring in the last period of a great number of affections of the brain, and especially of the spinal cord.

The eschars, if they but attain a certain extent, constitute, as you are aware, dangerous foci of infection; and, in fact, putrid intoxication, denoted by a more or less intense remittent fever, is one of the complications they most commonly provoke.

Next comes *purulent infection*, with production of metastatic abscesses in the viscera.[1] This species appears to be seldom met with.

We shall also notice *gangrenous emboli*. In this variety, thrombi impregnated with gangrenous ichor are transported to a distance and give rise to gangrenous metastases, which are principally observed in the lungs. This is a point upon which Dr. Ball and myself have insisted in a work published in 1857.[2] But long before us, and even long before the theory of embolism had been Germanized, M. Foville had expressed his opinion that a considerable number of cases of pulmonary gangrene, observed in the insane, and in different diseases of the nervous centres, are caused by "the transport into the lungs of a part of the fluid which bathes the eschars of the breech."[3]

The process of mortification tends gradually to invade the deeper tissues. The ruin that results is sometimes carried to the highest degree; thus the trochanteric serous bursæ may be laid open, the trochanter denuded of its periosteum, the muscles, the nerve-trunks, and arterial branches of a certain calibre laid bare. But the most dangerous accidents are those determined by the denudation and loss of substance of the sacrum and coccyx, the destruction of the sacro-coccygean ligament, and the consecutive opening of the sacral canal or arachnoid cavity. In consequence of these disorders, the pus and the gangrenous ichor may proceed to infiltrate the fatty cellular tissue which envelops the dura mater, or even, if this membrane be destroyed in any point, it may penetrate into the cavity of the arachnoid.[4]

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[1] Billroth und Wäckerling, in 'Langenbeck's Archiv für Klin. Chir.,' Bd. i, 1861, § 470. Fracture of the sixth dorsal vertebra, rapid formation of eschar on sacrum. Manifest symptoms of pyæmia: six or eight abscesses on the surface of the kidneys. Midderdorf, 'Knochenbrüch,' § 62. Fracture of the eighth dorsal vertebra. Rapid formation of eschar; pyæmia; metastatic abscesses in the lungs.

[2] "De la coïncidence des gangrènes viscerales et des affections gangréneuses extérieures, in 'Union Médicale,' 26 et 28 Janvier, 1860.

[3] 'Dictionnaire de Méd. et de Chir. Prat.,' t. i, p. 556.

[4] B. Brodie, *loc cit.*, p. 153. Velpeau, 'Anatom. Chirurgicale.' Ollivier (d'Angers) 'Traité des maladies de la moëlle épinière,' t. i, pp. 314, 324, 3d edi-

Under such circumstances, grave cerebro-spinal complications supervene; they may be collected into two principal classes. At one time we see a *simple purulent ascending meningitis;* at another, a sort of *ichorous ascending meningitis,* of which Lisfranc and Baillarger have reported many remarkable examples. In such a case, it is found that a puriform, grayish, acrid, and fetid liquid steeps the meninges and the cord itself, sometimes the lower part only is bathed in it, sometimes the whole cord. This liquid is occasionally found at the base of the encephalon, in the fourth ventricle, in the aqueduct of Sylvius, and even in the lateral ventricles. In all these points the cerebral matter is discoloured at its surface and to a certain depth, taking a slaty bluish tint which has several times been considered, but very wrongly, as constituting one of the characters of gangrene of the brain.[1] M. Baillarger was the first, I believe, to recognize the real nature of this alteration. What we have to note there is, above all, a phenomenon of imbibition, maceration, and *dyeing.* Remark that always, when ichorous cerebral meningitis has a sacral eschar as its starting point, the slaty tint is found throughout the whole extent of the spinal cord, it is constantly better marked there than in the encephalon, and more manifest the nearer you keep to the eschar. On the contrary, in the case where a sanious ulcer of the face, a cancroid for instance, after having destroyed the bone, has denuded the dura mater, the slaty coloration induced by ichorous maceration may, as I have many times observed, remain limited to the anterior lobes of the brain, in the regions corresponding to the bottom of the ulcer.

To these complications which I have been only able to indicate in a very summary manner, we must with Ollivier (d'Angers) connect the grave cerebral or cerebro-spinal symptoms, as yet but ill-defined, which rapidly terminate life in a great number of cases of disease of the spinal cord.

We have now to enter upon details and to show you the principal circumstances under which acute bed-sore is produced, under the influence of lesions of the brain and of the spinal cord, as well as the varieties of position and of evolution which it presents, according to the variety or seat of the lesion which has provoked its appearance. We shall also have to inquire whether the mode of production of this trophic lesion of the skin comes under the general theory which we have hitherto had to accept. With this aim, we shall successively review the different affections of the brain and of the cord which may give rise to acute bed-sore.

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tion, 1837. Moynier, "De l'eschare du sacrum et des accidents qui peuvent en resulter" ('Moniteur des Sciences Médicales et Pharmaceutiques,' Paris, 1859). Lisfranc, 'Archives Générales de Médecine,' 4e année, t. xiv, p. 291.

[1] Dubois (d'Amiens), 'Mémoires de l'Académie de Médecine,' t. xxvii, p. 50, 1865, 1866.

A. *Of acute bed-sore in apoplexy symptomatic of cerebral lesions in focal centres.* It is especially observed in the apoplexy consecutive on intra-encephalic hemorrhage, or on partial softening of the brain. But it may also be produced in meningeal hemorrhage, in pachymeningitis, and finally in cases when intra-cranial tumours give rise to apoplectiform attacks. The latter have often given me opportunities for observing it in patients attacked with partial encephalitis caused by wounds received in battle.[1]

The erythema, in all cases of this kind, usually shows itself from the second to the fourth day after the attack, rarely sooner, sometimes later. It affects a peculiar position. It is not in the sacral region, so commonly invaded in cases of spinal affection, that it develops, nor on any point of the median parts, but towards the centre of the gluteal region, and, most usually, if there be unilateral lesion of the brain, exclusively on the side corresponding with the hemiplegia (Fig. 3.)

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[1] The courtesy of my colleague, M. Cruveilhier, surgeon to La Salpêtrière, enables me to record the following fact, which I give as an example of the last-mentioned class.

The patient, Louis Ernst, a Saxon soldier, was picked up, at Villiers, on the field of battle, Nov. 30, 1870, and brought to the ambulance of La Salpêtrière, the same evening about nine o'clock. A bullet had traversed his skull, piercing it through and through; one of the orifices was situated on the upper part of the forehead, a little to the left of the median line; the other, on the right side, about the middle of the parietal bone. The cerebral substance protruded, like a mushroom, through the last-named orifice. The temporal region and the upper eyelid of the right side were ecchymosed and tumefied; profound coma. December 3d, somnolence; the patient, when interrogated sharply, mutters some inarticulate sounds; he puts out the tongue perfectly, when told; deglutition proceeds with ease. Almost complete hemiplegia is found to exist, with flaccidity of the muscles of the members of the right side. From time to time, without provocation, a sort of spasmodic contraction occurs in the superior member of this side, causing momentary pronation of the arm. The diaphragm seems to be also, from time to time, the seat of analogous contractions. The respiration, irregular at times, is calm, without stertor. There is no deviation of head, or eyes. The labial commissures are not drawn to one side. Sensibility appears much blunted over all parts of the body. No vomiting. Pulse very frequent, 140. December 4th (fifth day), same state as the previous day, but the somnolence is more intense than yesterday; contractions of the facial muscles are induced with difficulty, on forcibly pinching the skin. Involuntary passage of urine and feces. Skin warm, covered with perspiration; axillary temperature 41° C. *The commencement of an eschar is observed on the right gluteal eminence (the paralyzed side); nothing of the kind exists on the left. On the inner surface of the right thigh, a little above the knee, on a point where the flexed left knee seems to have exercised a rather prolonged pressure during the night, a bulla is found about the size of an almond, full of a lemon-coloured liquor and surrounded by an erythematous zone, of little extent.* The left knee, in the part where the pressure must have been, shows no trace of erythema or of epidermic elevation. The patient succumbed on the 5th December.

Autopsy.—The two cerebral hemispheres, at their middle and superior parts, in the points corresponding to the internal extremities of the anterior and posterior marginal convolutions, are transformed into a confused mess, partly bluish (slate-coloured). On a transverse section it is found that the softening extends to the centrum ovale (majus) of Vieussens, to the vicinity of the lateral ventricles, which, however, it does not attain, even on the left side, where the focus of the encephalitis is much more extensive, in all directions, than on the right. The optic thalami and corpora striata are perfectly normal. In the vicinity of the softened parts of the brain, the dura mater is covered with a neomembrane, of fibrinous character, and purulent in parts. The cranium is found to be fractured in several parts, in the neighbourhood of the orifices which gave passage to the projectile.

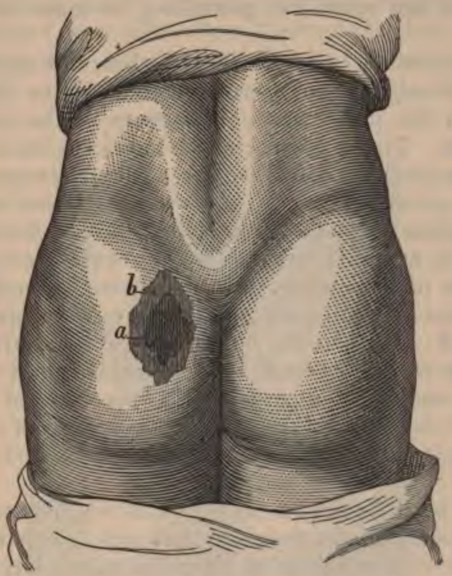

FIG. 3.—Gluteal eschar or the paralyzed side in a case of apoplexy, consecutive on hemorrhage: *a*, mortified portion; *b*, erythematous zone.

On the morrow or after-morrow, the bullous eruption and then the echymotic blotch make their appearance on the central part of the erythematous patch, that is, about two inches from the intergluteal fissure, and about an inch and a half beneath a supposititious line, drawn from its upper extremity, perpendicularly to its direction. Next, mortification of the derm supervenes in this same point, and it rapidly spreads, if the patient survive; but it is rather rare, on the whole, for the acute bed-sore of apoplectic sufferers to reach the stage of confirmed eschar.

It is likewise uncommon to observe, in addition to the gluteal eruption, bullæ or vesicles developed on the heel, the internal surface of the knee, and, in short, on the several points of the paralyzed lower extremity which may be subjected to a slight pressure.

I should not omit to point out to you that, according to my observations, this skin-affection appears but very exceptionally in cases which are to have a favourable termination; its appearance therefore constitutes a most inauspicious sign. We might, in fact, call it *decubitus ominosus*, or *ominous bed-sore*, by way of distinction.

This symptom, I repeat, is rarely deceptive, and as its existence may be discerned from the first days, it consequently acquires, as you will understand, a great value in doubtful cases. The very marked lowering of the central temperature, beneath the normal rate, observable at the outset of an attack, is to my knowledge the only sign that can rival the preceding, in cases of sudden hemiplegia.

The circumstances in which acute bed-sore of apoplectic patients develops, evidently do not permit us to refer to the intervention of pressure on the parts where it appears, as the only element in its production. The pressure is the same on both nates, but the eruption is exclusively produced, or at least always predominates in that of the paralyzed side. Many a time I was careful to make the patient repose upon the non-paralyzed side, during the greater part of the day, and this precaution has not in any way modified the production of the eschar. Besides, what, in such a case, could be the influence of a pressure which is only in operation for two or three days? Nor can the irritating contact of urine be given as the cause. In several cases, I have had this liquid drawn off hour by hour, day and night, during the whole time of the disease, in order to avoid as much as possible the irritation of the skin of the seat, and in spite of every care, the eschar was produced in accordance with the rules I have indicated.

What may be the organic cause of this singular trophic lesion? I was long under the impression that this lesion should be considered as one of the effects of neuro-paralytic hyperæmia, which betrays itself always, in a more or less prominent manner, you are aware, in members struck with hemiplegia of cerebral origin, by a comparative elevation of temperature. But this hypothesis is, as we shall see, open to a number of objections. The facts which will be set forth, as we proceed, render it probable that we must here recognize the irritation of certain regions of the encephalon, which, in the normal state, are believed to exercise a more or less direct influence over the nutrition of different parts of the external tegument.

B. *Of acute bed-sore of spinal origin.* When acute bed-sore appears under the influence of a lesion of the spinal cord, it shows itself in the very great majority of cases in the sacral region—and consequently above and internal to the chosen seat of eschars of cerebral origin. Here it occupies the median line and extends symmetrically, on either side, towards the adjacent parts. (Fig. 4.) It may, indeed, happen that only one side will be affected—in the case, for instance, where a lateral half of the cord is alone engaged, and then the cutaneous lesion frequently shows itself on the opposite side of the body from the spinal lesion.

The influence of attitudes here plays an important part. Thus it is customary when the patients are so placed as to repose on the side, during part of the day, to find, besides the sacral eschar, vast necrosive ulcerations developing on the trochanteric regions. It is also common enough to see, contrary to what happens in cerebral cases, that the different parts of the paralyzed limbs which are exposed to even slight and brief pressure, as the ankles, heels, and inner surface of knees, present lesions characteristic of acute bed-sore. Eschars may also show themselves, but indeed very rarely, on a level with the apex of the scapula, or over the olecranon process.[1]

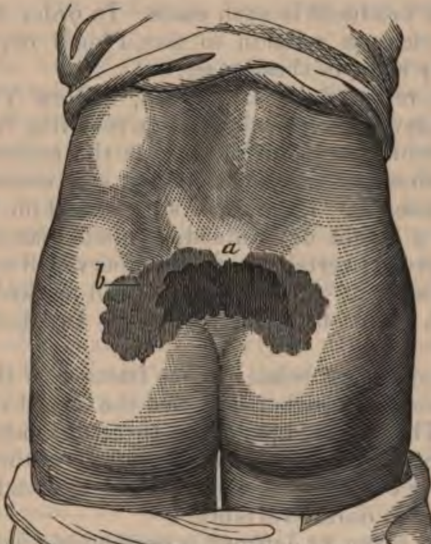

FIG. 4.—Eschar of the sacral region in a case of partial myelitis occupying the dorsal region of the spinal cord: *a*, mortified portion; *b*, erythematous zone.

Speaking generally, we may say that the spinal lesions which produce acute bed-sore are also those which give rise to rapid muscular atrophy and to other disorders of the same class. The almost simultaneous development of these different consecutive affections makes it seem probable, already, that they have a common origin.

It behooves us to remark, however, that this rule is far from being absolute. As a matter of fact, it is a characteristic of certain spinal affections that rapid muscular atrophy is developed without being accompanied by eschars; whilst there are others, on the contrary, where the eschars may be produced without the nutrition of the muscles in the paralyzed limb being affected. This is, in truth, a fact of great interest from the standpoint of pathological physiology, and one which we shall take care to bring into prominence (Fig. 4).

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[1] W. Clapp, 'Provinc. Med. and Surg. Journal,' 1851, p. 322, and Gurlt, *loc. cit.* p. 110, No. 76.

(*a*) We will mention, in the first place, the traumatic lesions of the spinal cord, those in particular which result from fractures or luxations of the vertebral column. Numerous cases of this kind, recorded by Bright,[1] Brodie,[2] Jeffreys,[3] Ollivier (d'Angers),[4] Laugier,[5] Gurlt,[6] and some others,[7] show with what rapidity sacral eschars may be produced in such cases. In order to enable you to form distinct ideas, in relation to this, I shall request permission to relate briefly some of these cases.

In one case, reported by Dr. Wood, of New York,[8] there was fracture of the seventh cervical vertebra, resulting from a fall down stairs; death took place four days after the accident. From the second day, redness of the sacral region was noticed, and a bulla formed at the coccyx. Hæmaturia supervened on the third day.

A fall from a height determined complete diastasis of the sixth and seventh cervical vertebræ; death supervened sixty hours after the accident, and, at that period, a well-marked bed-sore was already visible. This fact is recorded by Dr. Buchner, of Darmstadt.[9]

One of Jeffrey's cases relates to the fracture of the fourth dorsal vertebra; a confirmed eschar occupied the sacral region, from the fourth day. The eschar supervened three days after the accident, in a patient whose history has been narrated by Ollivier (d'Angers), on the authority of Guersant, and who had received a bullet in the body of the eighth dorsal vertebra.

Another case, given by Jeffreys, is particularly worthy of interest. The patient had fallen, from a ladder, a height of twenty-five feet. On post-mortem examination it was found that the bodies of the seventh and eighth dorsal vertebræ were broken in several pieces, and had been much displaced. On the day of the fall, the skin was cold, and the pulse barely perceptible. All the parts below the fracture were deprived of sensibility and motion. Next day, there were continual erections; "then supervened phlyctenæ in the region of the sacrum," and, on the same day, "the patient recovered his sensibility." I point out this last feature to your attention, because many authors have endeavoured, very erroneously, as you see, to make anæsthesia play an important part in the pathogeny of acute bed-sore of spinal origin. The persistence of sensibility, in the parts situated below the lesion, is also marked out, in a more or less explicit manner, in a case recorded by Colliny,[1] relating to a fracture of the seventh cervical vertebra, where the eschar appeared on the fourth day, as well as in a case mentioned by Ollivier (d'Angers),[2] where there was fracture of the twelfth dorsal vertebra. The eschar, in the latter case, made its appearance on the thirteenth day.

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[1] R. Bright, 'Report of Medical Cases,' t. ii, pp. 380, 432, London, 1821.

[2] B Brodie, 'Medic.-Chirurg. Transactions,' p. 148, t. ii, 1836.

[3] Jeffreys, "Cases of fractured spine," in 'London Med. and Surg. Journ.,' July, 1826.

[4] Ollivier (d'Angers), *loc. cit.*, t. i.

[5] Laugier "Des Lésions traumatiques de la moëlle épiniere," 'Thèse de concours,' Paris, 1848.

[6] E. Gurlt, 'Handbuch der Lehre von den Knochenbruchen,' 2 Th. i. Liefer Hamm. 1864.

[7] See an interesting chapter on this subject in Herr Samuel's work, *loc. cit.*, p. 239.

[8] Gurlt, *loc. cit.*, Tableau No. 97.

[9] Gurlt, *loc. cit.*, No. 86.

It is useless to multiply these examples, for all surgeons agree in acknowledging that the rapid formation of eschars is one of the most common of the phenomena consecutive on spinal lesions resulting from fracture with displacement of vertebræ. According to Gurlt, whose opinion as regards this subject is based on the study of a very large number of observations,[3] it is from the fourth to the fifth day after the accident that the first symptoms of acute bed-sore most usually commence to appear; but they may, as we have just seen, set in much earlier, as on the second day, and even sooner. It seems, and the remark has been made by Brodie, that the production of eschars occurs early in proportion as the lesion affects a high point of the cord. On the other hand, it would result from the statistics drawn up by J. Ashhurst that nutritive troubles become frequent in proportion as the wound is lower down. Thus, according to this author, eschars were only observed in three cases, after lesions of the cervical region (being $\frac{1}{41}$ per cent.); twelve times (or $\frac{9}{23}$ per cent.) for the dorsal region, whilst as regards the lumbar region, the proportion rose to 12 per cent. (seven cases).[4]

Priapism, clonic convulsions of variable intensity, supervening in the paralyzed members, either spontaneously or induced, tonic convulsions coming on in paroxysms—all those symptoms, which usually reveal a state of irritation of the cord and meninges, have been many times mentioned among the phenomena which, in fractures of the vertebral column, precede, accompany, or closely follow the precocious formation of eschars.

In such circumstances, as we have already seen, anæsthesia, of the parts smitten with motor-paralysis, is not a constant fact. As to the remarkable elevation of temperature of which these parts sometimes become the seat in consequence of vaso-motor paralysis,[1] it cannot now be ascertained whether it was then present or not, the attention of the observers not having been drawn to this particular phenomenon. We shall note, on the contrary, as a symptom which shows itself frequently at the same time as the acute bed-sore, the emission of sanguinolent urine, alkaline in reaction, and sometimes purulent. This is a fact to which we shall have occasion to revert. Necroscopical examination, hitherto, has not, in general, revealed anything in connection with spinal lesions which can be considered peculiar to the cases where rapidly developing eschars are produced. We frequently, however, find mention made of alterations of the spinal cord, which place beyond doubt the existence of an inflammatory process; the presence of purulent infiltration, and even the formation of abscesses in the midst of the softened parts, have been observed in several instances.

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[1] Quoted by Ollivier (d'Angers), *loc. cit.*

[2] Sensibility was also preserved in Dr. Büchner's case, quoted above, where the eschar appeared before the close of the third day.

[3] See Gurlt, *loc. cit.*, p. 94, analysis of 270 cases.

[4] J. Ashhurst, "Injuries of the Spine, with analysis of nearly 400 cases," Philadelphia, 1867.

b. The study of cases of hemiparaplegia, consecutive on wounds involving only a lateral half of the spinal cord, may furnish useful information concerning the pathogeny of acute bed-sore, and of some other trophic disorders of spinal origin. We learn, from the experiments of M. Brown-Séquard, that, after wounds of this kind, there supervenes in animals motor-paralysis of the lower extremity, on the same side with the lesion. The limb presents also a more or less marked degree of exaltation of tactile sensibility, and it likewise offers a notable elevation of temperature correlated with vaso-motor paralysis. The opposite limb preserves, on the contrary, its normal temperature and power of motion, whilst the tactile sensibility is much lessened, and may even be extinct. All these particulars are exactly reproduced in man under analogous circumstances. In his case, as in that of animals, we may also find different trophic derangements supervening, which appear almost simultaneously, and which are all manifestly due to spinal lesion. Among the nutritive lesions of this kind observed in man, we would especially point out the rapid diminution of the (faradaic) electrical contractility of the muscles, soon followed by atrophy,—a particular form of arthropathy to which I shall refer in a few moments—and finally, acute bed-sore. It is a remarkable thing that, whilst the arthropathy and muscular atrophy are to be found in the limb on the same side with the lesion, the eschar seems to prefer, as we have already remarked, to show itself on the member of the opposite side, where it occupies the sacral region, and the gluteal, in the immediate neighbourhood of the former. This peculiar disposition of the eschar in relation to the seat of the spinal lesion is, according to what M. Brown-Séquard has told me, a constant fact in the case of animals; in man, it has already been several times observed.

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[1] In a case of fracture of the vertebral column in the dorsal region, observed by J. Hutchinson, on the second day after the accident, the temperature of the feet, taken at the inner ankles, rose (to 101° F., or) above 38° Cent. In the normal state, according to observations made in London Hospital, by Dr. Woodman, the thermometer placed between the two first toes gave an average of 27.5° C. (81.5° F.)., the maximum being 34.5° C. (94° F.), and the minimum 21.5° C. (70° F.). See J. Hutchinson, "On Fractures of the Spine," in 'London Hospital Reports,' t. iii, 1866, p. 363. See also H. Weber and Gull, in 'The Lancet,' Jan. 27, 1872, p. 117. Clinical Society of London. [See also Mr. J. W. Teale, "Case of Remarkable Elevation of Temperature" after injury of the spine, in a young lady, where 122° F. (50° C.) is stated to have been observed, 'Lancet,' 1875, p. 340; and J. Hutchinson, "On the Temperature and Circulation after Crushing of the Cervical Spinal Cord," 'Lancet,' 1875, pp. 714, 747.]. (S.)

As an example of the class, I shall briefly cite the following facts;

A man, aged twenty years, whose history has been related by M. Viguès,[1] received on the back of the thorax, between the ninth and tenth dorsal vertebræ, a sword cut which, to judge from the symptoms, injured the left lateral half of the spinal cord chiefly. Motor paralysis immediately ensued, which, at first affecting both the lower extremities, appeared from the next day to be almost entirely confined to the left leg. Hyperæsthesia is very manifest in the latter member; the right limb presents, on the contrary, a well-marked obnubilation of sensibility, whilst the power of motion has nearly quite returned. The symptoms showed rapid improvement up to the twelfth day after the accident; on that day it was remarked that, without perceptible cause, the *left* leg, still more sensitive than in the normal state, had increased in volume, and also that in the left knee-joint there had accumulated a quantity of fluid sufficient to keep the patella raised half an inch above the condyles. Two days later an eschar was observed occupying the *right* lateral part of the sacrum and right gluteal region.

The case recorded by MM. Joffroy and Salomon,[2] of one of Dr. Cusco's patients, which was recently communicated to the *Société de Biologie*, reproduces, as it were, the foregoing case, even in its smallest details. In the former, as in the latter, after a traumatic lesion affecting one lateral half of the cord in the dorsal region, we find motor paralysis supervening in the inferior extremity that corresponds to the injured side; this limb presents a notable augmentation of temperature—a fact not mentioned by Viguès, though probably present—and manifest hyperæsthesia; whilst the opposite limb, unharmed in its motor functions, offers a remarkable diminution of all kinds of sensibility whilst preserving the normal temperature. In addition, and this is the point which we desire to put especially forward, shortly after the accident, and without any appreciable cause, there supervened an arthropathy in the knee of the paralyzed limb, whilst, in the vicinity of the sacral region, the nates of the member deprived of sensibility but not paralyzed in motion, became the seat of an eschar.[1]

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[1] Brown-Séquard, 'Journal de la Physiologie,' &c., t. iii, p. 130, 1863.

[2] 'Gazette Médicale de Paris,' Nos. 6, 7, 8, 1872.

I take the following case from an interesting work by Herr W. Müller:[2] in this instance the arthropathy is not mentioned; on the other hand, we find mention of rapid wasting of the muscles of the paralyzed limb, preceded for several days by a well-marked diminution of faradaic contractility. In all other matters, Müller's observation is in conformity with those of MM. Viguès and Joffroy. The case is that of a woman, aged 21, who received a stab with a knife in the back, at the fourth dorsal vertebra, the weapon, as the autopsy demonstrated afterwards, had completely divided the left lateral half of the spinal cord, two millimetres above the third dorsal pair. On the very day of the accident complete paralysis and hyperæsthesia of the left lower extremity was observed; the opposite limb was anæsthetic, but not paralyzed. On the second day it was found that the muscles of the paralyzed member and those of the lower part of the abdomen gave no reaction under the influence of faradaic stimulation, whilst, in the homologous parts of the opposite side, the electrical contractility remained normal. On the eleventh day an eschar was formed, occupying the sacral region and extending to the right gluteal eminence. On the same day, it was remarked that the paralyzed limb had notably wasted away, measuring about two inches less in circumference than the anæsthetic member. Death occurred on the thirteenth day. On a post-mortem examination, the borders of the spinal wound appeared tumefied, and of a reddish-brown colour; a thin purulent layer covered it. Below the wound the left lateral column, throughout its whole length, offered the anatomical characteristics of descending myelitis.

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[1] On account of the interest connected with it, we shall mention the principal details of this case.

The patient, Martin, aged about 40 years, was stabbed with a poignard, in the night of the 15-16th February, 1871. The weapon entered at the third dorsal vertebra. The direction of the wound is downward, backward, and to the right. Having been brought to hospital immediately after the wound, it was observed that, even then, the left inferior extremity was completely stricken with motor-paralysis, whilst the corresponding member on the other side showed nothing of the kind. February 16th, in the morning, the following note was made: Left lower extremity, complete motor-paralysis. The limb is perfectly flaccid; no trace of contraction, or rigidity, no spasmodic movements, nor subsultus. On the contrary, sensibility appears in the same limb to be exaggerated in all its modes; the least touch of the skin, especially near the foot, causes pain. Pressure has the same effect. A slight pinch or a tickle is followed by very painful sensations. Finally, the contact of a cold surface also produces painful sensations which the patient compares to those producible by a series of prickings. Right lower extremity: all the voluntary movements are perfectly normal, but *per contra*, the sensibility is almost completely destroyed. Complete analgesia; sensitiveness to touch almost null. The contact of a cold body is marked by an obscure dull prickling sensation. The insensibility is not limited, on the right, to the lower limb; it ascends to a level with the nipple. The urine and feces passed involuntarily.

February 24th (eighth day).—The same phenomena are observed; in addition it is noted that the left (motor-paralyzed) limb is warmer than the right. The patient complains of a sensation of constriction or rather of compression at the base of the thorax.

March 5th (seventeenth day).—The patient complains of troubled sight: the left pupil is more contracted than the right, and the vessels of the left eye are more voluminous and more numerous than those of the right eye. The evacuations have again become voluntary, for two days past. The state of the lower extremities is still unchanged.

March 13th (twenty-fifth day).—The right nates, since yesterday, has been the seat of vivid redness, and the epidermis has already fallen off from a part of the erythematous patch.

March 14th.—The derm is denuded to the size of a crown-piece on the right nates, near the sacrum: it is also ecchymosed (*acute bed-sore*). On Feb. 24th, it had been already remarked that some pain was felt when the left knee (motor-paralyzed limb) was moved; to-day, it is noted that this joint is swollen and red, and that it is, besides, the seat of spontaneous pains, exaggerated on movement (spinal arthropathy).

March 24th.—An ulceration, this day covered with granulations, has formed on the right nates, on a level with the ecchymosed patch. The swelling, redness, and pains have almost completely disappeared from the left knee.

[2] W. Müller, "Beiträge zur pathologisch Anatomie und Physiologie des menschlichen Rückenmarkes," Leipzig, 1871. Obs. i.

The simultaneous appearance of different trophic disorders noted in these cases, and in some others of the same kind, seem to indicate a common cause. This cause, to all appearance, is nothing other than the extension to certain regions of the inferior segment of the cord, of the inflammatory action originally set up in the immediate vicinity of the wound.[1]

That being admitted, it would seem legitimate, relying on the facts stated in the preceding lecture, to assign the rapid and general atrophy of the paralyzed muscles, noted in Herr Müller's case, to the invasion of the anterior cornu of the gray substance throughout the whole extent of the cord, whence nerves are given forth to the paralyzed muscles; the invasion in question taking place either progressively by direct downward propagation; or indirectly by the lateral columns. This lesion of the anterior cornu we shall mention, in a moment, to explain the development of the arthropathy described in the observations of Viguès and Joffroy. Now, with respect to the eschars, their appearance on the side opposite the spinal lesion tends to establish that the nerve-fibres (whose alteration, under such circumstances, provokes the mortification of the external tegument) do not follow the same course as those which influence the nutrition of joints and muscles, and that they, on the contrary, decussate in the cord in the same manner as the nerve-fibres subserving the transmission of tactile impressions.

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[1] In a work, recently published, I have endeavoured to establish that, after wounds of the spinal cord, irritative lesions such as hypertrophy of the axis-cylinders, proliferation of myelocytes, &c., may be observed at some distance from the spinal wound, above and below it, scarcely twenty-four hours after the accident. Charcot, "Sur la tumefaction des cellules nerveuses, motrices, et des cylindres d'axe des tubes nerveux dans certains cas de myélite," in 'Archives de Physiologie,' No. 1, 1872, p. 95. Obs. i.

Another item of information which we get from cases of hemiparaplegia consecutive on a unilateral lesion of the spinal cord, is this, namely: acute bed-sore may show itself independently of all neuroparalytic hyperæmia, since we observe it forming upon that side of the body where the vasor-motor nerves are not affected.

c. I shall now mention the case where myelitis results, not, as in the preceding instance, from a wound or attrition of the spinal cord, but from indirect traumatic influence, such for example as an effort made in raising a weight. Acute bed-sore may, in cases of this kind, be produced as rapidly as though there had been fracture of the vertebral column, as the following fact recorded by Dr. Gull demonstrates:

A man, aged 25, by trade a labourer in the London Docks, felt, after lifting a load, a sudden pain in his back. He was able to walk to his home, about a mile off. On the morning of the second day after, his lower limbs were completely paralyzed; two days later, or four days after the accident, an eschar had begun to form on the sacral region, and the urine which flowed from the bladder was ammoniacal. The patient succumbed ten days after paralysis had set in. At the post-mortem examination, it was noted, after careful scrutiny, that the bones and ligaments of the vertebral column presented no lesion; in the neighbourhood of the fifth and sixth dorsal vertebræ the spinal cord was transformed throughout its whole breadth into a thick liquid, muco-purulent in appearance and in colour both brown and greenish.[1]

Following the example of traumatic myelites, spontaneous acute myelitis also very frequently determines the precocious formation of sacral eschars, principally when it sets in suddenly, and when the evolution is rapid. In order not to enter on lengthy details, in connection with this matter, I shall confine myself to indicating some examples illustrative of this class of cases. The sore has been noticed on the fifth day in a case reported by Mr. Duckworth,[2] on the sixth day in the case of a patient under the care of M. Woillez, which M. Joffroy has communicated to me; on the ninth day in an observation of M. Engelken, on the twelfth day in another case related by the same author;[3] finally, in a case of a cervico-dorsal meningo-myelitis, published by MM. Voisin and Cornil, the eschar formed on the sixth day.[4] These examples might be easily increased.

Acute bed-sore frequently accompanies hæmatomyelia (which indeed appears to be, at least in a certain number of cases, only an accident of central myelitis); thus we found it in the case of Duriau, already quoted, where mortification showed itself in the sacral region only four days after the appearance of the first symptoms.[1]

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[1] W. Gull, "Cases of Paraplegia," in 'Guy's Hospital Reports,' 1858, p. 189, Case xxii.

[2] 'The Lancet,' 6 Nov., 1869, p. 638.

[3] *Loc. cit.*, 'Pathologie der acuten Myelitis,' Zurich, 1867.

[4] 'Gazette des Hôpitaux,' 1865, No. 26.

We may also observe rapid mortification of the skin of the sacral region supervening, even in spinal diseases of slow evolution, when a new course of active irritation intervenes on a sudden, or when acute inflammatory action is suddenly superadded to the initial lesion. Not only the exacerbation of partial sclerosed myelitis, but the sudden irruption into the rachidian cavity of pus emanating from an abscess, in the case of a patient suffering from vertebral disease may, as I can attest, determine the rapid formation of eschars. The same result would be likewise produced in case a tumour occupying the central portions of the cord should, by its presence, provoke the development of acute myelitis. Several examples of this kind are on record.[2]

If the evidences which we have collected here do not yet allow us to construct a pathogenic theory of acute bed-sore of spinal origin, they at least suffice, if I mistake not, to exhibit the principal conditions of the phenomenon. Manifestly, we must relegate to a secondary position the influence of pressure; and also that of vaso-motor paralysis which may be completely absent, as we have seen in relation to the hemiparaplegia resulting from the traumatic lesion of a lateral half of the cord. On the whole, the dominant and ever present fact is the active irritation of a more or less extensive region of the spinal cord—mostly showing itself, anatomically, by the characteristics of acute or superacute myelitis, and, clinically, by the assemblage of symptoms which are assignable to this kind of lesion. To explain the production of trophic disorders which issue in sacral mortification, here again it is not to absence of nerve-action that we should appeal, but to irritation of the spinal cord. This conclusion is in conformity with the experimental results which show that, in animals, the development of gangrenous ulcerations over the sacrum do not supervene on ordinary sections of the cord, but only in cases where inflammation has been set up in the neighbourhood of the spinal lesion.

It is scarcely probable that all the constituent parts of the cord are indiscriminately apt, under the influence of irritation, to provoke the development of acute bed-sore. The great frequency of this accident in cases of hæmatomyelia, and of acute central myelitis, where the lesion occupies chiefly the central regions of the spinal cord, seems to designate the gray substance as playing a predominant part in this respect. And this power is no doubt shared in by the posterior white fasciculi, for we know that the irritation of certain parts of these fasciculi has the effect of determining the production not only of different cutaneous eruptions, but also, though rarely indeed, that of dermal necrosis.[1]

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[1] 'Union Médicale,' t. i, 1858. p. 308.

[2] Amongst others see MacDowel's "Case of Paraplegia," in 'Dublin Quarterly Journal,' 1862.

On the other hand, it is perfectly established that all portions of the gray matter should not be indifferently accused; some of them, in fact may, as we have already suggested, undergo the gravest lesions, without acute bed-sore ever supervening. Such are the anterior cornua, whose lesions, *per contra*, have, as you are aware, a most decided influence on the nutrition of muscles and, as we shall soon see, on that of joints also.

Hence it is that sacral eschar is often absent in infantile spinal paralysis, and in adult spinal paralysis—diseases which are characterized anatomically by acute inflammatory lesions, systematically limited to the area of the anterior cornua; whereas, those other diseases, which affect the skin, depend upon irritative lesions occupying, either the central and posterior portions of the gray matter, or the posterior white fasciculi. From this particular point of view there is reason to recognize, in the spinal cord, the existence of two regions endowed with very distinct properties. Now, since these regions may be affected either separately or simultaneously, it follows that, in clinical practice, acute bed-sore and acute muscular atrophy will sometimes appear separately, and that they will sometimes, on the contrary, coexist in the same individual.

From what precedes, the influence of irritative lesions of the spinal cord upon the development of acute bed-sore seems to us placed beyond doubt. Herr Samuel has, however, advanced a contrary opinion; he thinks that the spinal cord does not play any part herein, and that the spinal ganglia or peripheral nerves are alone implicated. We shall mention elsewhere the arguments on which this theory is based; but we are now able to point out that it is in formal contradiction with what has been noted in the numerous cases of traumatic myelitis affecting an elevated part of the cord—the cervical region, for instance, or the superior portion of the dorsal region—cases where acute bed-sore supervenes in the sacral region, and certainly without the direct participation of the spinal ganglia, or of the peripheral nerves. The cases of hæmatomyelia, or of spontaneous central myelitis, followed by precocious eschars, are likewise adverse to the views of Herr Samuel.

It is not alleged, however, that the irritative lesions of the peripheral nerves, and perhaps also those of the spinal ganglia, may not sometimes have the effect of determining the rapid formation of eschars. No doubt, the examples published of dermal necrosis developed in consequence of a puncture, incomplete section, or compression of a nerve are rare enough; but many of them are thoroughly convincing.[2] In connection with this, I will relate the case of a female patient which I have been recently studying at La Salpêtrière. She had, on the left side, an enormous fibrous tumour which compressed, in the pelvis, the roots of the ischiatic and crural nerves of the corresponding lower extremity. There had resulted a paretic state of this member, accompanied by acute pains running along the track of the principal nerve-trunks. One morning, shortly after the appearance of the first symptoms of compression, it was remarked that an eschar had rapidly formed near and to the left of the sacral region. The inner surface of the left knee, likewise, in a spot which had been pressed upon by the right knee for a considerable time during the night, in consequence of the attitude of the patient when asleep, presented some pemphigoid bullæ, full of a brownish liquid, which soon gave place to an eschar. Nothing of the kind was developed on the right knee. This is perhaps the place to mention that spontaneous zona which, in certain cases at least, is very probably connected with the inflammation of some nerve, may, according to the remark of Rayer,[1] occasionally issue in the more or less deep mortification of the skin. I have been often a witness to this fact, occurring among the aged persons in this hospital, and I have been many times able to satisfy myself that pressure on the spot occupied by the eruption did not here play an essential part. As to acute bed-sore of the breech, I am much inclined to believe that, in a certain number of cases, it should be attributed to an irritative lesion of the nerves of the cauda equina. A case recently published by M. Couyba, in his inaugural dissertation, may be cited as one of several examples of this class.[2]

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[1] See *ante*, Lecture iii, § 1, p. 52.

[2] See amongst recent published facts, a case reported by Dr. W. A. Lanson

III.

On Arthropathies of Cerebral or Spinal Origin.—Nutritive disorders consecutive on lesions of the nervous centres not unfrequently take up their seat in the articulations. The varieties presented

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('The Lancet,' 30 Dec., 1871, p. 913), and two cases of Dr. Vitrac ('Union Médicale de la Gironde,' t. ii, p. 127, and 'Revue Phot. des Hôpitaux,' 1871).

[1] Rayer, 'Maladies de la Peau,' t. i, p. 335.

[2] A young private in the Garde Mobile received a bullet wound, at the outposts of Clamart. The projectile had entered near the anterior extremity of the tenth rib on the left side, and had emerged on the right side of the vertebral column, about three inches from the spine, on a level with the second lumbar vertebra. Paresis, with acute hyperæsthesia of the lower extremities, followed. On the right gluteal eminence a bulla (which quickly gave place to an eschar) appeared on the fifth day after the accident. The eschar extended in a progressive manner, so as at last to cover the whole of the sacro-gluteal region. Death occurred on the nineteenth day.

Post-mortem.—A purulent mass covers the anterior and posterior surfaces of the cord, and extends from the cauda equina to the cervical region. The cord itself when examined, first, in the fresh state, next in numerous hardened sections, did not exhibit any alteration. On the other hand, a certain number of nerve-tubes in the nerve-filaments which form the cauda equina presented the anatomical characters of fatty granular degeneration.—Couyba, 'These de Paris,' 1871. Obs. xiii, p. 53.

by these articular affections, according to the nature of the cerebral or spinal lesions from which they arise, have led me to establish two principal categories.

A. The first comprises arthropathies of acute or subacute form, accompanied by tumefaction, redness, and sometimes by pain of a more or less severe character. This form was indicated for the first time, if I mistake not, by an American physician, Professor Mitchell,[1] who observed it in the paraplegia connected with Pott's disease of the vertebræ, in which, however, it is very rare, in my opinion.[2] It happens more frequently as a consequence of a traumatic lesion of the spinal cord, as we find from the sufficient evidence of the cases, above quoted, which have been recorded by MM. Viguès and Joffroy.[3] A case of concussion of the cord, related by Dr. Gull, supplies an analogous demonstration.[4]

Acute or subacute inflammation of the joints of paralyzed limbs may supervene also, in *spontaneous myelitis;* as examples of this class I may mention a case reported by Dr. Gull,[5] and another case which M. Moynier published in the "Moniteur des Sciences Médicales" for 1859. The second case relates to a young man, aged eighteen, who, after lodging for a long time in a damp place, and undergoing great fatigue, had presented all the symptoms of subacute myelitis. Paralysis of motion began to show itself on the 25th of January; it became complete on the 9th February. On the 23d of the same month, the skin of the sacral region presented an erythematous patch which gave place to an eschar, on the 5th of March. On the 6th of this month, there was severe pain in the right knee, which was swollen, and in which the sensation of fluctuation was perceptible. In addition, there was painful tumefaction of the tibiotarsal articulation of the same side. On the 9th of March, the knee had decreased in size, and on the same day, eschars made their appearance on the heels. The autopsy revealed a focus of ramollissement situated not quite two inches above the cauda equina.

Finally, in a case of central myelitis in a child, having its origin in the neighbourhood of a solitary tubercle situated in the cervical region of the cord, Dr. Gull records the formation of an intra-articular effusion, occupying one of the knees, at the time when the paralysis began to invade the lower extremities.[6]

It is remarkable to see these arthropathies, consecutive on the different acute and subacute forms of myelitis, frequently forming, when the muscles of the paralyzed limbs are beginning to waste away, or again when an eschar is being rapidly developed on the breech.

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[1] Mitchell, "American Journal of the Medical Sciences," t. viii. p. 55, 1831.

[2] I have, however, seen one knee become the seat of a subacute arthropathy in a woman suffering from paralysis consecutive on Pott's disease. This case has been recorded in the thesis of M. Michaud, "Sur la méningite et la myélite dans le mal vertébral," Paris, 1871.

[3] *Loc. cit.*, pp. 91, 92.

[4] Gull, "Guy's Hospital Reports," 3d series, t. iv, 1858. Case xxvii.

[5] Gull, *idem*, Obs. xxvii.

[6] Gull, *loc. cit.*, Case xxii.

The *arthropathy of hemiplegic patients*, first described I believe in 1846, by Scott Alison,[1] afterwards by Brown-Séquard, and the

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[1] Scott Alison, "Arthrites occurring in the Course of Paralysis," Note read before the Medical Society of London, Jan. 16, 1846, 'The Lancet,' t. i. p. 276, 1846. It is manifestly to the arthritis of hemiplegic patients, such as we have described it ('Arch. de Physiologie,' t. i), that the note of Dr. Alison refers. It is a characteristic of the affection to remain confined to the paralyzed limbs, and not to extend to the sound members. The affected joints are hot, swollen, and in some cases painful, either spontaneously or on movement made. The parts most frequently affected are the knee, elbow, wrist, hand, and foot. This form of arthritis seems to show itself chiefly in cases where the hemiplegia is consecutive on encephalitis or on brain softening. Two cases, selected from a number of others of the same kind, and cited as examples, deserve to be briefly recorded here:—

Case I.—A woman, aged 49 years, who had long enjoyed perfect health and had never suffered from any form of arthritic disease, was suddenly struck with hemiplegia; some days after, tumefaction and heat at the wrist of the paralyzed side set in, and a little later on, the knee and foot of the same side became swollen and painful in their turn. There was no œdema. The paralyzed limbs were rather rigid.

On *post-mortem* examination, partial softening of the brain was discovered. Each renal pelvis was filled with little calculi of uric acid.

Case II.—A man, aged 54, house painter, who had experienced several attacks of gout, was struck with sudden hemiplegia. Soon after the wrist, the hand, and the foot, became hot and swollen. The paralyzed limbs were rigid.

At the autopsy, the brain appeared softened, and a voluminous blood-clot was found in one of the lateral ventricles.

Dr. Alison endeavoured to explain the occurrence of arthritis in the course of (hemiplegic) paralysis, by showing that "the healthy relation between the living tissues and the materials of the blood was disturbed. Two morbid conditions gave rise to this disturbance, viz., a state of reduced vitality in the paralyzed parts, and the presence of exciting and noxious agents in the blood. In proof of this various facts were referred to, and the author related two singular cases of the inflammatory red line of the gums following the use of mercury, in paralysis of one side of the face, being strictly confined to the paralyzed side of the mouth. The paralyzed parts were in fact more delicate tests of poisons than parts in a state of health. In proof of the presence of exciting agents in the blood the gouty diathesis of the second case and the lithic acid calculi in the pelvis of the kidney of the first case, were adduced."

We, in our turn, would point out that, most certainly, these cases are altogether exceptional, as regards the question at issue, for most frequently, as may be understood from a perusal of the cases published in our work ('Archives de Physiologie,' t. i.) the arthritis supervenes in hemiplegic patients as a more or less direct consequence of the cerebral lesion, quite apart from all influence of gout, rheumatism, or other diethetic condition.

Hence, whilst acknowledging the accuracy of Dr. Alison's clinical descriptions, I am unable to endorse the pathogenic theory which he has proposed. I am, however, far from denying that the articulations of paralyzed members, in cases of hemiplegia of cerebral origin, may, as Dr. Alison holds, be particularly disposed to become foci of elimination for other agents previously accumulated in the blood. I myself communicated to the Société de Biologie, at the time of its occurrence, a case in which this particular disposition was very prominent. A woman, aged about 40 years, had been suddenly struck with right hemiplegia, three years before her admission into my wards. The paralyzed limbs were strongly anatomical and clinical characters of which I have made known, belongs, if I mistake not, to the same category. In this second variety, as well as in the first, the arthropathies are limited to the paralyzed limbs and mostly occupy the upper extremities. They supervene, especially, after circumscribed cerebral ramollissement (*en foyer*), and, more rarely, as a consequence of intra-encephalic hemorrhage.

They usually form fifteen days or a month after the attack of apoplexy, that is to say, at the moment when the *tardy contracture* that lays hold on the paralyzed members appears, but they may also show themselves at a later epoch. The tumefaction, redness, and pain of the joints are sometimes marked enough to recall the corresponding phenomena of acute articular rheumatism. The ten-

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contractured now and again, the several joints of these limbs, the knee especially and the foot, were the seats of tumefaction and pain. The patient, being aphasic, in a high degree, it was impossible to ascertain if she had been previously subject to gout or rheumatism.

At the autopsy, we found a vast ochreous cicatrix, the vestige of a focus of cerebral hemorrhage, situated exterior to the extra-ventricular nucleus of the corpus striatum. In most of the articulations of the limbs on the right side, which had been hemiplegic, the diarthrodial cartilages were incrusted towards their central parts with deposits of urate of soda, both crystallized and amorphous. The joints of the limbs, on the other side, presented no similar appearance. Some white striæ, which were found on microscopical and microchemical examination to be formed by urate of soda, were noticed in the kidneys.

It is undoubtedly most remarkable to find, in this case, that the gouty deposit forms exclusively in the joints of the paralyzed members; but, I cannot too often repeat that facts of this kind are exceptional, and, in any case, they have nothing in common, from a pathogenic point of view, with the ordinary arthritis of hemiplegic patients ('Cas d'Hubert,' see Bourneville, 'Etudes cliniques et thermometriques sur les maladies du système nerveux,' p. 58).

The merit is due to M. Brown-Séquard of having directed attention anew to the arthropathy of hemiplegic patients, and of having determined the organic cause, better than Dr. Alison had done. He thus expresses himself in a lecture published in 'The Lancet' ("Lectures on the Mode and Origin of Symptoms of Diseases of the Brain," Lecture i, Part ii, 'The Lancet,' July 13, 1861). After having admitted that the painful sensations, such as formication and prickling, which are experienced in the paralyzed members, in consequence of a cerebral lesion, result generally from a direct irritation of the encephalic nerve-fibres, he adds:—

"It is most important not to confound these sensations (which are referred sensations, like those taking place when the ulnar nerve has been injured at the elbow joint) with other and sometimes very painful sensations in the muscles or in the joints of paralyzed limbs. These last sensations very rarely exist when the limbs are not moved, or when there is no pressure upon them; they appear at once, or are increased by any pressure or movement. They depend upon a subacute inflammation of the muscles or joints, which is often mistaken for a rheumatic affection. This subinflammation in paralyzed limbs is often the result of an irritation of the vaso-motor or nutrition nerves of the encephalon."

Before M. Brown-Séquard, and before even Mr. Scott Alison, many physicians had already remarked the arthritis of paralytic patients, but without bringing out the interest connected therewith. Consult R. Dann, 'The Lancet,' t. ii, p. 238, 1841. Durand-Fardel, 'Maladies des Vieillards,' p. 131. Paris, 1854, Observation, Lemoine. Valleix, 'Guide du Médecin Praticien,' t. iv, 1853, p. 514. Grisolle, 'Pathologie Interne,' 2nd édition, t. ii, p. 257.

dinous sheaths are, indeed, often affected at the same time as the articulations.

I have shown that we have here a true synovitis with vegetation, multiplication of the nuclear and fibroid elements which form the articular serous membrane, and augmentation in number and volume of the capillary vessels which are there distributed. In intense cases, a sero-fibrinous exudation is produced, with which are mingled, in various proportions, white blood-corpuscles that may become abundant enough to distend the synovial cavity. The diarthrodial cartilages and ligamentous parts have not hitherto appeared to present any concomitant lesion perceptible to the naked eye. On the other hand, the tendinous synovial sheaths, in the neighbourhood of the affected joints, take part in the inflammatory process, and appear greatly congested.[1]

It is needless to insist upon the interest which pertains to these arthropathies as regards diagnosis,—articular rheumatism, whether acute or subacute, being an affection often connected with certain forms of cerebral softening, and one which, indeed, shows itself also, occasionally, after traumatic causes capable of determining shock in the nervous centres. On the other hand, many affections of the spinal cord are erroneously attributed to a rheumatic diathesis in consequence of the co-existence of these articular symptoms. The clinical characters which render it easy to recognize arthropathies correlated with lesions of the nervous centres, and which allow them to be distinguished from cases of rheumatic arthritis, are chiefly these:

1st. Their limitation to the joints of the paralyzed members.

2d. The generally determinate epoch in which, in cases of sudden hemiplegia, they make their appearance on the morbid scene.

3d. The coexistence of other trophic troubles of the same order, such as eschars of rapid formation; and (when the spinal cord is involved) acute muscular atrophy of the paralyzed members, cystitis, nephritis, &c.

B. The type of the second group is to be found in progressive locomotor ataxia. Allow me to fix your attention for an instant upon this species of articular affection, in which I take a paternal interest, all the more lively because the signification I attached to it has had to encounter many sceptics. And at first, a word as to the clinical characters of the *arthropathy of ataxic patients.*[1]

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[1] Charcot, "Sur quelques arthropathies qui paraissent dépendre d'une lésion du cerveau ou de la moelle épinière," 'Archives de Physiologie,' t. i, p. 396, Pl. vi, figs. 1, 2, 3, 4, 5, 6. Paris, 1868. The arthropathy in question should apparently not be confounded with the articular affection which has been described, in latter days, by Herr Hitzig of Berlin, "Ueber eine bei schweren Hemiplegien, Auftretende Gallenkaffection," in 'Virchow's Archiv,' Bd. xlviii, hft. 3 u. 4, 1869. This species appears, especially, when the hemiplegia is of comparatively old date, and the patients have been able to walk for some time; it chiefly occupies the shoulder-joint, and results principally from the displacement of the articular surfaces, occasioned by the paralysis of the muscles which surround the joint.

This disorder generally shows itself at a determinate epoch of the ataxia, and its appearance coincides, so to speak, in many cases with the setting in of motor incoördination.

Without any appreciable external cause, we may see, between one day and the next, the development of a general and often enormous tumefaction of the member, most commonly without any pain whatever, or any febrile reaction. At the end of a few days the general tumefaction disappears, but a more or less considerable swelling of the joint remains, owing to the formation of hydarthus; and sometimes to the accumulation of liquid in the periarticular serous bursæ also. On puncture being made, a transparent lemon-coloured liquid has been frequently drawn from the joint.

One or two weeks after the invasion, sometimes much sooner, the existence of more or less marked cracking sounds may be noted, betraying the alteration of the articular surfaces which, at this period, is already profound.[2] The hydarthus becomes quickly resolved, leaving after it an extreme mobility in the joint. Hence consecutive luxations are frequently found, their production being largely aided by the wearing away of the heads of the bones which has taken place. I have several times observed a rapid wasting of the muscular masses of the members affected by the articular disorder.

Ataxic arthropathy usually occupies the knees, shoulders, and elbows; it may also take up its seat in the hip-joint The anatomo-pathological information which we possess respecting it, is as yet very imperfect. However, one character is apparently constant, namely, the enormous wearing down which is exhibited in a very short space of time by the articular extremities. At the end of three months, this head of a humerus which I show you, and which belonged to a female patient in whom we were enabled to study the invasion of the arthropathy, was, as you may remark (Fig. 5), to a great extent destroyed. I would call your attention to the fact, that you do not find on this specimen, the bony burr around the worn articular surface, which would not fail to be present if this were a case of common dry arthritis.[3]

I now place before you in order to establish the contrast, a knee-joint also taken from a woman who presented the symptoms of ataxic arthropathy, but in whom the articular affection was of much older date. Besides the wearing down of the articular surfaces which, as in the preceding case, is carried very far, you notice here the presence of foreign bodies, of bony stalactites, and, in a word, of all the customary accompaniments of *arthritis deformans*. These latter alterations, I repeat, were absolutely wanting in the first case. On this account, I am led to believe that they are nowise necessary, and that they are produced in an accidental manner, and to all appearance chiefly by the more or less energetic movements to which the patients sometimes continue to subject the affected members.

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[1] Charcot, "Sur quelques Arthropathies," &c., p. 1. 'Archives de Phys.,' t. i, 1868.

[2] In some cases the cracking sounds have preceded, by several days, the appearance of the general tumefaction of the member; but, as a rule, the latter is the first symptom observed.

[3] Compare Charcot, "Ataxie locomotrice progressive, arthropathie de l'épaule gauche. Résultats nécroscopiques," in 'Archives de Physiologie,' t. ii, p. 121, 1869.

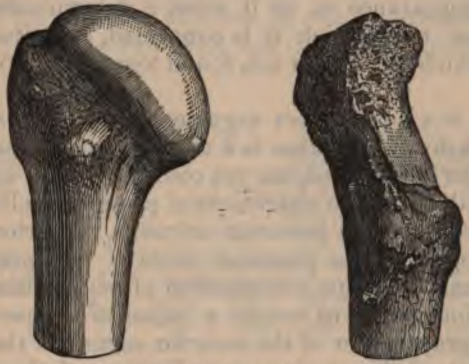

Fig. 5.—Upper extremity of a healthy humerus, and a humerus presenting the lesions of ataxic arthropathy.

I wish to confine myself at present to this indication of the most general features of the arthropathies of ataxic patients, for this is a subject which I propose to treat hereafter in more detail. What I have to say will suffice, I hope, to show that the articular affection in question is, itself also, the expression of trophic disorders directly dependent on the lesion of the spinal nerve-centre. But here are the principal arguments upon which I base my opinion.

I would point out, in the first place, the absence of all traumatic or diathetic cause of rheumatism or of gout, for instance, which might explain the appearance of the articular disease in the cases which I have studied. Herr R. Wolkmann[1] has said that the arthropathy of ataxic patients is simply the result of the distension of the articular ligaments and capsules, in consequence of the awkward manner of walking peculiar to this class of persons. The cases, which are now numerous, in which our arthropathy affected the upper extremities, and occupied either the shoulder or the elbow, are sufficient to prove that the interpretation proposed by Wolkmann could have but a very narrow bearing. The influence of a mere mechanical cause cannot be invoked, at least not as a principal agency, even in cases where the arthropathy occupies the lower extremities. I have, in fact, taken care to point out, supporting my words by oft repeated clinical observations, that the articular affection in question is developed at a comparatively early epoch of the sclerosis of the posterior columns, and at a time when motor incoördination is as yet null, or scarcely manifest.

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[1] Canstatt's 'Jahresbericht,' 1868-1869, 2 Bd., p. 391.

The clinical characters of our arthropathy are, besides, really special. Its sudden invasion, marked by the general tumefaction of the member; the rapid alterations of the articular surfaces; finally, its appearance at, as it were, a determinate epoch of the spinal disease with which it is connected, constitute so many peculiarities which are, if I err not, found together in no other articular affection.

But here is a more direct argument. Holding as we did that the arthropathy in question is a trophic lesion consecutive on the disease of the spinal cord, we yet could not think of connecting it with any of the common alterations of progressive locomotor ataxia —with sclerosis of the posterior columns, posterior spinal meningitis, or atrophy of the posterior roots of the spinal nerves. On the other hand, a minute examination of many cases had taught us that it was impossible to invoke a lesion of the peripheral nerves. It is in the gray matter of the anterior cornua of the cord that the starting point of this curious complication of the ataxia is to be found according to our belief.[1] It is not very rare to find the spinal gray matter affected in locomotor ataxia; but the lesion is then generally found in the posterior cornua. Now, it was quite different in two cases of locomotor ataxia, complicated with arthropathy, in which a careful examination of the cord has been made; the anterior cornua were, in both cases, remarkably wasted and deformed, and a certain number of the great nerve-cells, those of the external group especially, had decreased in size, or even disappeared altogether without leaving any vestiges. The alteration, besides, showed itself exclusively in the anterior cornu corresponding to the side on which the articular lesion was situated (Fig. 6). It affected the cervical region, in the first case, where the arthropathy occupied the shoulder; it was observed, a little above the lumbar region, in the second case which presented an example of arthropathy of the knee. Above and below these points, the gray matter of the anterior cornua appeared to be exempt from alteration.

It may be asked whether this alteration of one of the anterior cornua of the cord, which microscopical examination reveals, may not be a result of the functional inertia to which the corresponding member has been condemned on account of the articular lesion. This hypothesis must be rejected because, on the one hand, in both of our cases, the members affected by the arthropathies had preserved to a great degree their freedom of motion; and, on the other hand, the lesion of the gray matter differed essentially here from that which is produced after the amputation of a member, or the section of the nerves supplying it.

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[1] See Charcot et Joffroy, "Note sur une lesion de la substance grise de la moëlle épinière, observée dans un cas d'arthropathie liée à l ataxie locomotrice progressive," 'Archives de Physiologie,' t. iii, p. 306, 1870.

From what precedes, I hope to have made it appear at least highly probable that the inflammatory process, first developed in the posterior columns, by gradually extending to certain regions of the anterior cornua of the gray matter was able to occasion the development of the articular affection in our two patients. If the results obtained in these two cases are confirmed by new observations, we should be naturally led to admit that arthritic affections connected with myelitis, and those observed to follow on cerebral softening are likewise due to the invasion of the same regions of the gray matter of the spinal cord. In cases of brain-softening, the descending sclerosis of one of the lateral columns of the cord might be considered as the starting point of the diffusion of inflammatory work.

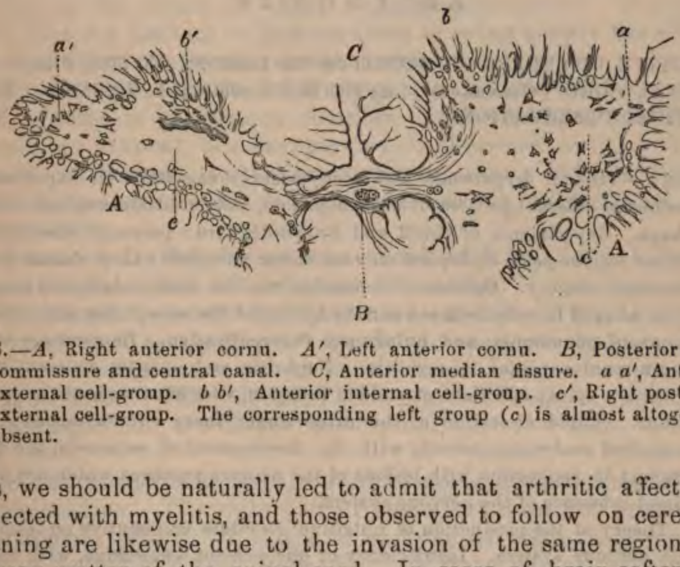

FIG. 6.—*A*, Right anterior cornu. *A′*, Left anterior cornu. *B*, Posterior gray commissure and central canal. *C*, Anterior median fissure. *a a′*, Anterior external cell-group. *b b′*, Anterior internal cell-group. *c′*, Right posterior external cell-group. The corresponding left group (*c*) is almost altogether absent.

MM. Patruban,[1] Remak,[2] and quite recently, Herr Rosenthal,[3] have observed in *progressive muscular atrophy*, arthropathies which by their clinical characters are closely allied with those of ataxic patients. This is nothing surprising, if we remember that a primary or secondary irritative lesion of the nerve-cells of the anterior cornua of the spinal gray matter appears, in the majority of cases, to be the starting-point of the amyotrophy which, in clinical practice, is usually designated by the name of progressive muscular atrophy.

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[1] Patruban, 'Zeitschrift für prakt. Heilkunde,' 1862, No. 1.

[2] Remak, 'Allgemeine medizinische central Zeitung,' March, 1853, 20 st.

[3] Rosenthal, 'Lehrbuch der Nervenkrankheiten,' p. 571. Wien, 1870. See also Benedikt, 'Elektrotherapie,' t. ii, p. 384.

For to-day, gentlemen, I shall stop here in this investigation, which I expect to bring to a conclusion at our next conference.

LECTURE IV.

NUTRITIVE DISORDERS CONSECUTIVE ON LESIONS OF THE BRAIN AND SPINAL CORD (*Conclusion*). AFFECTIONS OF THE VISCERA. THEORETICAL OBSERVATIONS.

SUMMARY.—Visceral hyperæmia and ecchymoses consecutive on experimental lesions of different portions of the encephalon, and on intra-encephalic hemorrhage. Experiments of Schiff and Brown-Séquard: personal observations. These lesions seem to depend on vaso-motor paralysis: they should form a separate category. Opinion of Schroeder van der Kolk, relative to the relations alleged to exist between certain lesions of the encephalon and different forms of pneumonia, and pulmonary tuberculization. Hemorrhage of the supra-renal capsules in myelitis. Nephritis and cystitis consecutive on irritative spinal affections of sudden invasion, whether traumatic or spontaneous. Rapid alteration of the urine under these circumstances; often remarked contemporaneously with the development of eschars in the sacral region; its connection with lesions of the urinary passages which are due to direct influence of the nervous system.

Theory of the production of nutritive disorders consecutive on lesions of the nervous system. Insufficiency of our present knowledge, with respect to this question. Paralysis of the vaso-motor nerves; consecutive hyperæmia; trophic disorders not produced. Exceptions to the rule. Irritation of the vaso-motor nerves: the consequent ischæmia seems to have no marked influence on local nutrition. Dilator and secretor nerves: researches of Ludwig and Claude Bernard; analogies between these two orders of nerves. Theoretical application of trophic nerves. Samuel's hypothesis. Exposition. Criticisms. Conclusion.

GENTLEMEN: The reverberation of lesions of the nervous system is not felt only in the peripheral parts, in the skin, bones, and muscles, the viscera themselves may also be influenced by these lesions. It is known that certain alterations of the encephalon especially those which affect the optic thalami, the corpus striata, and particularly the different parts of the isthmus, whether caused experimentally, or spontaneously produced, are occasionally followed by the manifestation of certain visceral lesions.

Thus in some experiments made by Professor Schiff[1] and by Brown-Séquard[2] there frequently supervened in the lungs, stomach, or kidneys, either simple hyperæmia or real ecchymoses, consequent on traumatic irritation of the optic thalami, corpora striata, pons Varolii, and bulbus rachidicus, etc. Again, nothing is more common, as I have shown, than to find in man, in cases of apoplexy symptomatic of cerebral softening, but especially in cases of intra-encephalic hemorrhage in foci, patches of congestion and real ecchymoses on the pleuræ, the endocardium, and the mucous membrane of the stomach.[3]

What is the reason of these singular alterations? Professor Schiff does not hesitate to look on them as being simply the effects of the paralysis of the vaso-motor nerves.

I am very much inclined, for my part, to believe that the pathogenic process is here more complex. Nevertheless, the direct influence, so to speak, of neuro-paralytic hyperæmia on the development of ecchymoses, in apoplectic patients, seems well established by the following case which I communicated to the *Société de Biologie*, in 1868.

A female in La Salpêtrière was struck with apoplexy, followed by hemiplegia of the left side, and succumbed a few days after. The paralyzed members had presented a comparatively considerable increase of temperature. At the autopsy, we discovered in the right hemisphere a recent hemorrhagic focus, occupying the corpus striatum. The epicranial aponeurosis presented on the left, or hemiplegic side, a wine-red colour, and, here and there, spots of ecchymosis.

The abnormal colour and the ecchymoses stopped suddenly at the median line. The right half of the epicranium had preserved its customary pallor: no traces of ecchymosis were to be found. Spots of ecchymosis were observed in the substance of the pleuræ, of the endocardium, and of the mucous membrane of the stomach.[4]

However it be, the visceral lesions in question differ by important characteristics from the affections which form the principal object of our studies. Those are congestions and ecchymoses, as we have said; the symptoms of inflammation are never superadded without the intervention of some accessory cause, a thing altogether unnecessary, as you are aware, in cases of common trophic lesions. We have consequently grounds for placing in a separate category, at least temporarily, the congestions and ecchymoses which make their appearance consecutively on lesions of different parts of the encephalon.

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[1] Schiff, 'Gazette Hebdomadaire,' t. i, p. 428. 'Lezioni di Fiziologia sperimentale sul systema nervoso encefalico,' pp. 287, 297, 373. Firenze, 1866. 'Leçons sur la Physiologie de la Digestion,' t. ii, p. 433. Florence, 1867.

[2] 'Société de Biologie,' 1870.

[3] 'Comptes Rendus de la Société de Biologie,' 19 Juin, 1869. Paris, 1870.

[4] Ibid., année 1868. Paris, 1869, p. 213.

Again, some authors, Schroeder van der Kolk amongst others, consider that the different forms of pneumonia, and even of pulmonary tuberculization, which frequently supervene in the course of certain encephalic affections, depend, in such circumstances, on the influence of lesions of the brain and medulla oblongata upon the lungs. But it must be acknowledged that the cases upon which the alleged connection rests are not yet sufficiently convincing.[1]

Spinal lesions, as well as lesions of the encephalon, may be followed by the production of visceral ecchymoses. It will suffice for me to remind you that if the lumbar cord be wounded with a puncturing instrument, in a guinea-pig, effusion of blood into the supra-renal capsules occasionally follows.[2] It seems right to recall this experiment of Brown Séquard, because human pathology supplies us with analogous facts. Quite recently my friend Dr. Bouchard has told me of a case of acute myelitis, observed in Professor Béhier's wards, and promptly terminating in death. At the autopsy, besides the lesions of partial myelitis, recent hemorrhagic foci were discerned in the substance of the supra-renal capsules.

But, I repeat, congestive and ecchymotic lesions appear to form a separate order. On the other hand, the affections of the kidneys and of the bladder, to which I wish now to call your attention, are, by the general bearing of their characteristics, allied to the group of trophic lesions, properly so called.

You are aware that *nephritis* and *cystitis* are very common complications of irritative spinal affections, of sudden invasion, whether they be of traumatic origin, or spontaneously developed.

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[1] Schroeder van der Kolk. "Atrophy of the brain," Sydenham Society, 1861. The author dwells on the fact that, according to the statistics published in his Treatise on the Spinal Cord, all the epileptic patients whose tongues were bitten, succumbed in consequence of phthisis, pneumonia, or marasmus. He adds that, according to Durand-Fardel, patients attacked by brain-softening almost always die of a pulmonary affection, and he quotes Engel's statistics which support this view ('Prager Vierteljahrschr.,' vii Jahrg.. Bd. iii). He refers to the experiment, now of old date, in which Schiff believed he saw, in the rabbit, tubercles (?) developed in the upper lobe of the lung after the section of the ganglion of the pneumogastric nerve ('Wunderlich's Archiv,' 6 Jahr., 8 heft, pp. 769 et seq.), and finally points out that, among the observations collected by Brown-Séquard in his "Recherches sur la Physiologie de la protubérance annulaire" ('Journal de la Physiologie,' t. i), there are a certain number where phthisis and pneumonia occasioned death. Cruveilhier, Andral, and Piorry had long since noted the prodominant part which, according to them, acute pneumonia plays in the issue of apoplexies determined by cerebral softening or hemorrhage.

According to the observations which I have collected at La Salpêtrière, lobular or lobar inflammations of the lungs would be less frequent, under the circumstances, than these physicians seem to believe.

[2] Brown Séquard, "Influence d'une partie de la moëlle épinière sur les capsules surrénales," 'Comptes Rendus de la Société de Biologie,' 1851, t. iii, p. 146.

It has been long recognized that, after fracture of the vertebral column with consecutive lesion of the spinal cord, the urine frequently undergoes rapid alteration. Dupuytren pointed out, as you may recollect, that in such circumstances the catheter left in the bladder to guard against retention of urine, became rapidly coated with a calcareous incrustation.[1] But it was Brodie especially, who called attention to the characters presented by the urine in the case of persons stricken with traumatic paraplegia.[2] On the eighth, on the third, and on the second day, he has observed the urine become alkaline, and exhale a fetid ammoniacal odour, at the moment of emission. Soon afterwards, it contained blood-clots, muco-pus, deposits of ammoniaco-magnesian phosphates. It would, in fact, be easy to gather from authors a very great number of cases in which the urine-changes, noticed by Brodie, have occurred in the first days following on paraplegia, determined by fracture of the vertebral column.[3] At the autopsy, in such cases, more or less advanced lesions of purulent nephro cystitis are found.[4]

But traumatic lesions of this kind are, in general, little suited to illustrate clearly the relations which exist between inflammation of the urinary passages, and alterations of the spinal cord. For it can always be supposed, if strictly considered, that a fall or concussion violent enough to produce fracture of the spine, may have determined the vesico-renal lesions by the same shock.

It is otherwise when we have to deal with an affection, spontaneously developed in the spinal cord, or with a wound determined in this organ by the blow of some sharp weapon. Now, even in cases of this kind, it is common to find, a short time after the invasion of the paralytic phenomena, a more or less marked modification in the constitution of the urine, connected with nephro-vesical alterations, not unfrequently of a serious character. I shall confine myself to mentioning, by way of example, the following facts.

In one case, previously described, of hemiparaplegia caused by a knife stab, the urine became alkaline on the third day, and soon after muco purulent. Death occurred on the thirteenth day.

At the autopsy, very evident inflammatory lesions were found in the kidneys, ureters, and bladder.[5] In an analogous case, reported by M. Brown-Séquard, on the authority of Dr. Maunder,[1] the urine was likewise found to be alkaline, a very short time after the accident. Cases of this kind are very interesting inasmuch as they show that a unilateral and very circumscribed lesion of the cord suffices to determine a more or less grave and generalized affection of the urinary passages.

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[1] Ollivier (d'Angers), *loc. cit.*, t. i, p. 372.

[2] Brodie, 'Medico-Chirurg. Transactions,' *loc. cit.*

[3] See Stanley, 1st case. Urine strongly ammoniacal on the fifth day; 2d case, ammoniacal urine on the fourth day. 'London Medico Chirurg. Trans.' t. xviii, p. 1. Jeffreys: urine ammoniacal and sanguineous, the seventh day (Ollivier, d'Angers, *loc. cit.*, t. i, p. 322).

[4] Molendrinski, "Bruch des Zweiten Lendenwirbels," Langenbeck's 'Archiv,' xi Bd., 1869, p. 859.

[5] Case of W. Müller, see *ante*, 'Third Lecture,' p. 70.

Alike in spontaneous acute myelitis, of sudden invasion, and in hæmatomyelia, is the appearance of ammoniacal, sanguineous, and muco-purulent urine a fact of frequent occurrence, soon after the manifestation of paralytic symptoms. Thus the urine was already greatly altered on the fifth day, in the case of acute myelitis, which we have quoted from Dr. Duckworth;[2] on the sixth day, in that given by M. Joffroy.[3] It was ammoniacal the fourth day, in Dr. Gull's patient;[4] sanguineous the third, and purulent the ninth, in a case recorded by Herr Mannkopf.[5]

In the case of hæmatomyelia, recorded by M. Duriau,[6] the urine was ammoniacal and contained blood clots the fourth day; it presented the same character the sixth day and became gradually purulent in a case reported by Ollivier (d'Angers) on the authority of Monod.[7] In this instance, there was hemiparaplegia, consecutive on the presence of a hemorrhagic focus occupying a lateral half of the spinal cord. You will find, in the work of M. Rayer, the description of lesions, frequently grave, affecting the kidneys, the renal pelves, and the bladder, to which these changes in the urine should be attributed.[8]

Many of the observations, just cited, contain an item of information the importance of which cannot escape your notice. It is mentioned that the urine which until then was normal became, as I have said, ammoniacal, sanguineous, or muco-purulent, at the very time when eschars were being developed on the sacral region, and when the electrical contractility was beginning to grow feeble in the paralyzed muscles.[9]

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[1] 'Journal de Physiologie,' t. vi, p. 152, 1863.

[2] See *ante*, 'Third Lecture,' p. 72.

[3] *Idem*, p. 72.

[4] *Idem*, p. 72.

[5] 'Berliner Klin. Wochenschrift,' t. i, No. 1, 1864.

[6] 'Third Lecture,' p. 72.

[7] Ollivier (d'Angers), *loc. cit.*, t. ii, p. 177.

[8] Rayer, 'Traité des maladies des reins,' t. i, p. 530 et seq. "According to my observations," Rayer writes, "in the diseases of the spinal cord, when the urine contained in the bladder is alkaline, it is so, not because of a decomposition difficult to be explained without atmospheric contact, and in *a short space of time*, but rather by a vice of renal secretion which should be attributed, in most cases, to *an inflammatory irritation of these organs*."

As regards the description of alterations in the urinary passages, consecutive on acute affections of the spinal cord, consult, Engelken, *loc. cit.*, p. 12. Mannkopf, 'Bericht über die Versammlung zu Hannover,' p. 259; and 'Berlin. Klin. Woch.,' t. i. Compare, Rosenstein, 'Nierenkrankheiten,' 2 Ed., p. 287. Berlin, 1870.

[9] Ollivier (d'Angers) had already remarked that, in traumatic paraplegia, when the urine alters at an early period the eschars are found to form rapidly in the sacral region *Loc. cit.*, t. ii, p. 37.

How are we to understand so rapid a development of the inflammatory lesions of the urinary passages after acute affections, spontaneous or traumatic, of the spinal cord? Manifestly, the paralytic retention of the urine cannot here be pleaded, at least not as the sole, nor even as the predominant, pathogenic element. Neither is it possible to attach great weight to the opinion[1] which would attribute the urine-changes, in such circumstances, to the introduction of unclean catheters, carrying vibriones. In point of fact, the introduction of vibriones into the bladder could only be a chance occurrence, whilst the appearance of ammoniacal, sanguineous, and purulent urine, in the course of acute myelitis is, like the production of eschars, what may be termed a regular fact.

The notorious insufficiency of the pathogenic conditions just enumerated, renders it at least highly probable that there is a direct action of the nervous system engaged in the production of the affection of the urinary passages which we are considering. The cause of this affection, as of the other trophic lesions which often show themselves at the same time, would therefore be the irritation of certain portions of the spinal centre, and more particularly, no doubt, of the gray substance.

THEORETICAL PORTION.

GENTLEMEN: In the foregoing series of studies, we have often had occasion to acknowledge that the development of the trophic disorders, ensuing after lesions of the nervous system, is not in general (contrary to a wide-spread opinion) the consequence of absence of action of different parts of that system. Far from that, these affections would result, in our view, from the irritation set up, under certain conditions, either in the peripheral nerves or in the nervous centres themselves. Thus, we find ourselves possessing a view, which is of primary importance to the pathologist, and you, without further explanation, can readily divine the practical deductions to which it may guide us.

But it must next be ackowledged that this wholly empirical notion marks only the first step taken towards the scientific knowledge of the phenomena, which observation has allowed us to establish. For, if we know the mode of initial alteration and its seat as well, there remains yet to be determined, in the first place, the means by which this lesion reacts upon the peripheral parts.

Evidently, this reaction is produced by means of the nerves, but that also, from a theoretical point of view, is an insufficient datum. It is necessary to endeavour to be more precise, and to seek what is the element, in that physiologically complex totality called a nerve, by which the transmission is operated, and also the mechanism of this transmission.

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[1] Traube, 'Munk. Berliner Klin. Wochensch.,' p. 19, 1864.

I approach the question just raised, with an almost absolute certainty of not being able to answer it by rigorous arguments. Perhaps, I should have avoided it, desirous of not wasting your valuable time, if I were not convinced that it behooves us at least to show the inanity of a theory which professes to resolve it, and which to-day enjoys an almost uncontested sway.

You are not unaware, gentlemen, of the considerable influence which has been attributed to the vaso-motor nerves in the explanation of pathological phenomena. I am far from wishing to ignore the fact that a goodly number of these phenomena do, indeed, directly depend either on the dilatation or on the contraction of the smaller vessels, determined by nervous influence. But in so far as the trophic disorders which form the object of our studies are concerned, I hope that it will not be difficult to show, from a brief examination, that the *vaso motor* theory is altogether insufficient.

In order to attain this aim, I am induced to remind you of some of the experimental facts which have unveiled the functions of these centrifugal nerves whose ultimate ramifications go to animate the muscular coat of the smaller vessels. I shall, in the first place, recall the phenomena noticed when these nerves have been paralyzed in consequence of complete section, for instance.

Section of the vaso-motor nerves[1] has the immediate effect of producing a paralytic dilatation of the vessels to which they are distributed. Hence results a state of hyperæmia, termed *neuro-paralytic*, which has been especially well studied in cases of section of the great sympathetic nerve in the cervical region, but which is to be found with almost identical characters after a great number of lesions of the nervous centres or of the peripheral nerves. The consequences of this hyperæmia are, from our point of view, particularly worthy of interest. You know that the part answering to the divided nerve, presents a relative elevation of temperature, which appears solely to result from the afflux of a greater quantity of blood. You know that throughout the whole extent of the hyperæmic territory an exaltation seems also to ensue of all the vital properties of every element and every tissue. At least, the sensitive as well as the motor nerves, and the muscles themselves become more excitable,[2] and the latter preserve, longer than is usual after death has occurred, their proper contractility.[3] Nevertheless, in spite of these new conditions—and this is a point which requires to be set prominently forth—the accomplishment of the intimate acts of nutrition appears to be modified in nothing essential. Thus, in the experiments of M. Ollier,[1] agreeing in that respect with those of M. Claude Bernard, there is not found to supervene, in young animals after section of the great sympathetic in the neck, either an acceleration or an exaggeration in the growth of the parts of the face, even when subjected for months to neuro-paralytic hyperæmia. Nor does it appear that this hyperæmia, however intense or prolonged it may be, has ever the effect, save under exceptional circumstances to be hereafter mentioned, of determining by itself the development of inflammatory action. And if the experimenter intervenes and applies agents capable of provoking inflammation, the morbid process determined by this influence goes through its course in the hyperæmic parts as if under normal conditions; it offers no special characters, except, indeed, that the injured parts tend to heal with greater promptness.

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[1] For the physiology and pathology of the vaso-motor nerves, consult, Vulpian, "Leçons sur l'appareil vaso-moteur," recueillies par C. Carville, Paris, 1875 (*Note to the second edition*).

[2] Brown-Séquard, 'Lectures on Physiology and Pathology,' Philadelphia, 1860, p. 1457.

[3] Brown-Séquard, *loc. cit.* Joseph, in 'Centralblatt,' 1871, No. 46.

It is true that, in reference to the latter points, M. Schiff professes a very different opinion. He affirms, in fact, that changes of nutrition originate in the hyperæmic parts, in cases of vaso-motor paralysis, under the influence of the slightest local mechanical irritant,[2] and that inflammation here readily takes on a destructive character.[3] But upon this subject he is in direct opposition to MM. Snellen, Virchow,[4] and O. Weber.[5]

In a recent experiment, besides, Herr Sinitzin states that after the extirpation of the superior cervical ganglion on one side, the introduction of a slender piece of glass into the cornea of the same side caused merely a very slight inflammatory reaction, sometimes scarcely noticeable; whilst on the opposite side, in the selfsame animal, its introduction caused, on the contrary, a most active inflammation with purulent infiltration of the cornea, iritis, panophthalmia, etc.[6] M. Claude Bernard, indeed, long since pointed out that ablation of the superior cervical ganglion appears to retard the manifestation of the nutritive disorders occasionally determined in the eye by section of the fifth pair of nerves,[7] and Herr Sinitzin has arrived at the same results in his experiments.

From this you may perceive that, contrary to the opinion of Professor Schiff, neuro-paralytic hyperæmia does not create in the parts it occupies, a peculiar predisposition to the production of trophic derangement. It would even seem that these parts are better able to resist the action of disorganizing causes and that any disorder set up there is more speedily repaired than elsewhere.

In man, so far as this question is concerned, little difference is to be found occurring, from what is observed in animals. At all events, neuro-paralytic hyperæmia has been seen to persist for a long period in parts of the body, as for instance in the face, without any nutritive disorder ever supervening. M. Perroud has collected a certain number of cases of this kind, in a memoir read in 1864, before the Medical Society of Lyons. It suffices indeed, to glance at the numerous works which, of late years, have been published upon *Angioneuroses* to perceive that nutritive disorders are a rather rare accompaniment of neuro-paralytic hyperæmia. A new argument may, perhaps, be added in support of the thesis which we uphold. It is this: The elevation of temperature, tested by means of a thermometer, is, we have said, a phenomenon indissolubly linked with the existence of partial hyperæmias of neuro-paralytic origin. This local hyperthermia should necessarily exist in parts presenting the trophic derangements we have described, if these were really dependent on a neuro-paralytic cause. Now that does not happen, as a general fact. If a marked elevation of temperature has been many times observed in those regions of the body where an eruption of zona, consecutive on neuralgia, or neuritis had developed;[1] on the other hand, it may be said that irritative lesions of the peripheral nerves, in the conditions when they usually determine trophic disorders, appear to be accompanied rather by a lowering of the thermal standard than by its elevation. This lowering has been observed at every period of the nerve-affection; it has been noted near the commencement,[2] still oftener in the advanced stages.[3] When spinal lesions are concerned, it is true that occasionally the members subject to trophic troubles, rapid muscular atrophy, bullar eruptions, or eschars, exhibit a more or less marked elevation of temperature.[4] But at other times, perhaps in the majority of instances, this phenomenon is absent; thus it is absent in partial myelitis,[5] and in infantile paralysis;[6]—the same rule holds good for cases of slow evolution, such as, for instance, progressive muscular atrophy.[1]

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[1] Ollier, 'Journal de la Physiologie,' t. vi, p. 108.
[2] Schiff, 'Physiologie de la digestion,' p. 235, t. i. 'Lezioni di Fisiologia,' Firenze, 1866, p. 35.
[3] Schiff, 'Digestion,' t. ii, p. 423.
[4] Virchow, 'Cell-pathologie,' 4 ed., p. 158.
[5] O. Weber, 'Centralblatt,' 1864, p. 148.
[6] Sinitzin, 'Centralblatt,' 1871, p. 161.
[7] Claude Bernard, 'Système Nerveux,' t. ii, p. 65, 1865.

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[1] Horner, quoted by O. Wyss, 'Archiv der Heilkunde,' 1871. See note to p. 563. Charcot, 'Neuralgie du nerf cubital. Eruption du Zona sur le trajet du nerf affecté; examen thermométrique,' Thèse de Mougeot, Paris, 1867, p. 101.

[2] Folet, "Cas de Contusion du plexus brachial, observé par M. Lannelongue," 'Etude sur la température des parties paralysées,' Paris, 1867, p. 7.

[3] Hutchinson, *loc. cit.* Earle, "Medico-Chirurg. Trans.," vol. vii, 1816, p. 173. Yellowly, *id.*, t. iii. W. B. Woodman, in 'Sydenham Society's Transactions.' Translation of Wunderlich, 'On Temperature in Diseases,' p. 152. S. W. Mitchell 'Injuries of Nerves,' Philadelphia, 1872, p. 175. In two cases of nerve-wounds with "glossy skin," the region occupied by the trophic lesion was from one to two degrees warmer than the corresponding region of the healthy limb. But above this point, the thermometer marked one degree lower than on the healthy limb. H. Fischer, "Ueber trophische Störungen nach Nervenverletzungen an den Extremitäten," in 'Berliner Klin. Wochenschr..' 1871, No. 13. The temperature of the limbs, on which the most varied trophic disorders occur, is, at first, higher than that of the healthy members, afterwards it is relatively lower; but there are many exceptions to this rule.

[4] Levier, "Cas d'Hematomyélie," *loc. cit.*

[5] Mannkoff, *loc. cit.*

[6] Duchenne (de Boulogne), *loc. cit.*, 3d edition, p. 398.

You observe, from what precedes, that the trophic disorders connected with irritative lesions of the nervous centres may, in a considerable number of cases at least, occur without that elevation of temperature which should, I repeat, be necessarily present in all cases, if they really originate in hyperæmia, consecutive on paralysis of the vaso-motor nerves.

Hence it follows that neuro-paralytic hyperæmia and the production of trophic derangement are, in ordinary conditions, phenomena independent of each other. But as we suggested, a little ago, there are circumstances in which, contrary to the usual rule, local nutrition may receive a serious blow from the mere fact that the part has been withdrawn from vaso-motor innervation. This happens, as experiments attest, when the whole organism has been subjected to potent debilitating causes. Thus a vigorous animal has long had the great sympathetic nerve divided on one side of the neck; nevertheless, no injury has been experienced in the parts corresponding to the distribution of the divided nerve. But let the animal fall sick, or be deprived of food, then the scene changes immediately, and we see, says M. Claude Bernard, inflammatory phenomena ensue in that side of the face which corresponds with the experimental section. On that side, even without the intervention of any external agent whatever, the conjunctiva and the pituitary membrane rapidly begin to suppurate.[2]

It is legitimate to suppose that the animals in which Professor Schiff saw trophic lesions supervene, consecutively on neuro-paralytic hyperæmia, under the influence of the slightest mechanical irritation, had been suffering from the debilitating conditions noticed by M. Claude Bernard. In man, the same concurrence of circumstances ought necessarily to determine effects analogous to those observed in animals, and we may, indeed, question whether some of our trophic derangements are not really produced in this manner. Such is, perhaps, the case as regards the *acute bed-sore* of apoplectic patients. Here, in fact, the general condition is most unfavourable, and the gluteal eschar occupies precisely that side of the body which on account of the motor paralysis, presents a relative elevation of temperature, evidently connected with vaso-motor hyperæmia.[3]

However it be, this pathogenic interpretation can have but a very restricted application, for acute bed-sore arising from lesion of the nervous centres may appear in many cases, after hemi-lateral lesions of the spinal cord for example,[1] on parts of the body where the vaso-motor innervation is not visibly affected and apart from every symptom indicative of great depression of the organism.

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[1] Landois und Mosler, in 'Berliner Klinisch. Wochenschr.,' 1868, s. 45. For examples of depressed temperature supervening after spinal injury, see J. Hutchinson: "Temperature, etc., after crushing of the cervical spinal cord," 'Lancet,' pp. 713, 747. 1875. (S.)

[2] Claude Bernard, 'Physiologie du Système Nerveux,' t. ii, p. 535, Paris, 1858. 'Medical Times and Gazette,' p. 79, t. ii. 1861.

[3] See *ante*, Third Lecture, p. 62.

We have now to inquire whether the irritation of the vaso-motor nerves can account for the phenomena which are not explained by the paralysis of the same nerves. Let us first take experimental irritation. Partial ischæmia, of a more or less intense character, is the most prominent result of this irritation: it may be carried so far that not even a drop of blood flows on pricking the skin.[2] The parts, in which vascular spasm thus impedes the circulation, grow pale and cold; their vital activity decreases; the excitability of the muscles and of the nerves falls below the normal standard.[3] It is natural to think that grave nutritive lesions, tending to necrobiosis or to sphacelus, should necessarily result from the prolongation of such a state. But it is important to observe that this is commonly a question of a temporary phenomenon, lasting at longest for a few hours only. For by the very fact of the prolongation of the irritation the action of the nerve seems to exhaust itself, and hyperæmia, generally, soon follows on anæmia.[4] However, by reproducing, at short intervals, the irritation of the vaso-motor nerves, it is possible to cause the ischæmic state to predominate for a certain time. Still I do not believe that any trophic lesion would be ever experimentally produced, by this method. Herr O. Weber, who, by means of an ingenious apparatus, says he has kept up, for nearly a week, irritation of the cervical sympathetic nerve, of a permanent character, so to speak, and marked by a thermal decrease of 2° C., has not seen the slightest trace of nutritive trouble supervening in the corresponding side of the face.[5] Cases connected with human pathology yield the same testimony. Thus it is not rare to find, in certain cases of *angioneuroses*, amongst hysterical patients for example, a very intense and very persistent partial ischæmia; yet trophic troubles never show themselves, under such circumstances.[6] As to the instances of spontaneous gangrene, which have been attributed to vascular spasm, they would not have, to judge from my own observations, the signification assigned them; for, in all cases of this kind which I have happened to meet with, I have found the calibre of the vessel occluded by an alteration of the arterial walls, or obstructed by a thrombus.[1]

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[1] See *ante*, p. 72.

[2] Brown-Séquard, 'Course of Lectures,' &c., p. 147, Philadelphia.

[3] Brown-Séquard, *loc. cit.*, p. 142.

[4] Waller, 'Proc. Royal Society, London,' vol. ii, 1860–72, p. 89 et seq.

[5] O. Weber, 'Centralblatt,' No. 10, 1864, p. 147.

[6] Liégeois, 'Société de Biologie,' 1859, p. 274. Charcot, in 'Mouvement Médical,' Nos. 25, 26, 1re série; No. 1, nouvelle série, 1872.

From the foregoing observations you perceive that it is neither to a paralytic nor to an irritative affection of the vaso-motor nerves, *properly so called*, that we should attribute the trophic disorders which supervene in consequence of lesions of the nervous system.

Physiological experiments, in these latter years, have revealed the existence of centrifugal nervous filaments, the irritation of which has the effect of determining dilatation of the bloodvessels, and consequently hyperæmia of the region to which these nerves are distributed.

Whilst irritation of the common vaso-motor nerves produces ischæmia, irritation of the *dilator nerves* determines, on the contrary, a more or less intense hyperæmia. The chorda tympani may be considered, at the present moment, as the prototype of dilator nerves. But nerves endowed with similar properties exist in the face,[2] in the penis,[3] and in the abdomen.[4] There are probably others in existence also in many parts of the body.

We are far from possessing a certain knowledge of the mode of action of these nerves. According to the hypothesis adopted by M. Claude Bernard, this is how we should explain the remarkable afflux of arterial blood which takes place in the submaxillary gland, under the influence of the chorda tympani. In the opinion of that eminent physiologist, the irritation of this nerve is transmitted to the little ganglionic masses which are distributed in great numbers on the intra-glandular extremities of the nerve. These would, in their turn, react by a sort of *nervous interference*[5] on the nerve-filaments of the great sympathetic or vaso-constrictor, and paralyze their action. Thus the chorda tympani, and the same doubtless should be said of all the other dilator nerves, would play the part of a check nerve in relation to the vaso-motors. Hence, as you see, the result of the action of the dilator nerves would, according to this theory, be simply vaso-motor paralysis.[6] Now, if it be true

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[1] See the Thesis of M. Benni, 'Recherches sur quelques points de la gangrène spontanée,' Paris, 1867. Obs. v, xi, xvii.

[2] Claude Bernard, 'Revue Scientifique,' t. ii, 2 serie, 1872. Schiff, 'Digestion,' t. i, p. 252.

[3] Erector nerves of Eckhardt, 'Beitrage zur Anat. und Phys.,' t. ii. Löven, 'Bericht der Sachs. Ges.,' 1866.

[4] Claude Bernard, *loc cit.*

[5] Claude Bernard, *loc. cit.*, p. 1204.

[6] For a clinical illustration in the human subject of the physiological theory, see "Note sur la Paralysie vaso-motrice généralisée des membres superieurs," par le Dr. Sigerson (Publications du 'Progrès Médical'), 1874, Adrien Delahaye, Paris; or Translation by Dr. Barnard Ellis, New York.

The following are some of the principal features of this case, to which Dr. Duchenne (de Boulogne) invited the writer's attention, and which, at the request of that eminent physician, was made the subject of a detailed study, from pathological and physiological stand-points. The extract is taken from Dr. Barnard Ellis's translation:—

"*History.*—The patient C—, aged 50, a copper-trimmer by trade, is a man of that vaso-motor paralysis, even when carried very far, as happens for instance in cases of complete section of the vaso-motor nerves,

robust constitution, and florid complexion, who has hitherto enjoyed excellent health. He has had neither cough, nor colic, nor any of the symptoms usually assigned to copper-poisoning, whether the heart, the respiratory, or the digestive organs be considered. The hands, which are in an abnormal condition, present no lesion except the cicatrix of an old whitlow on the left forefinger. He came to be treated for impotence, and that, at first, was all he complained of; but other phenomena were soon discovered, some of which were traced back several years. By careful questioning the following facts were elicited. In 1872 he was aware of a weakness in the arms and legs, but most especially in the knees. This sensation, however, neither became localized nor remained constant; it seemed to flit through all his members. In 1873, he noticed that it predominated in the left knee. This uneasy sensation, which gave no pain, and was transient, seemed to ascend along the leg from the calf to the thigh; and the proof that it was not merely a subjective sensation lies in the fact that the weakness of the leg increased so much at times that he was obliged to sit down. He usually recovered, however, in a few minutes, and was able to go about his work as before. No aggravation of his symptoms occurred when he walked out; on the contrary, the exercise did him good, and after a brisk walk of half an hour he felt a marked sensation of pleasurable ease.

This disorder, as we see, was intermittent, showing itself after intervals of comparative health. In January last (1874), however, he was attacked, in a more enduring manner, in both upper and lower extremities,—the feeling of debility being greatest in the left arm and right leg. So much was he enfeebled that, whereas when formerly attacked he could lift a weight of about two or three pounds, he became at this time unable even to keep his forearm flexed on his arm. He preserved the power of flexion, but not the power of maintaining it, as, in a few seconds, the forearm would fall of its own weight. At this stage, the muscular force of the hands, tested by the dynamometer of Dr. Duchenne (de Boulogne), was equivalent, on an average, to 43 kilogrammes, or 94.6 lbs.

The colour of his hands had become a deep red, and this florid flush extended up the forearms, gradually diminishing in intensity. Let us add that, notwithstanding the vascular disturbance, there was nothing that could be referred to the existence of scleroderma, to which there was some superficial resemblance. The patient complained of great heat in the hands and forearms, and this increase of temperature was plainly perceptible to all who touched them; and it is a very remarkable fact that their sensibility was so greatly augmented, that everything he touched—instruments, wood, or paper—appeared to him as cold as ice. He was troubled with formication in the forearms, which increased to a painful degree when he rubbed his hands together as when washing them. Heat aggravated and cold diminished the pain,—facts of which he had become aware on using cold and warm water.

As to the inferior extremities, they presented different phenomena. There was, indeed, debility, as has been remarked, but the symptoms of the disease seem to have decussated. Whilst the left arm was the weaker, the right leg was the more feeble. Instead of the hyperæsthesia which we observed in the hands, there was a notable loss of sensibility in the right foot, so that he did not feel the ground when walking. This foot seemed to him asleep or benumbed. There was, at times, slight formication in the right leg, but very little in the left. Nor was there a hypothermal condition here, as in the upper extremities. Although the temperature of the soles of his feet seemed normal to himself, during the daytime, it had been remarked that, when he was lying down and during the night, they were ice-cold to the touch.

In the lumbar region, he had experienced an intense itchiness, as if he had been beaten with nettles. This unpleasant sensation was not constant, and had only appeared five or six times in all, and then only in the morning and at night, when he was dressing or undressing and exposed to the cold air—phenomena occasionally present in the case of persons suffering from urticaria. There were is not a cause of trophic disorders, the same rule must plainly hold good as regards the paralysis produced under the influence of the dilator nerves. But, gentlemen, as you will see further on, the mode of action of the dilator nerves may be considered from an altogether different point of view.

I would remind you of the fundamental experiments of Ludwig, relative to the influence of certain nerves on the secretion of the submaxillary gland.[1] Notwithstanding the criticisms which have assailed the conclusions drawn from his experiments by this celebrated physiologist, these conclusions do not appear to have been shaken. I have to request your permission to enter into some details in reference to this subject; they are absolutely necessary for the object we have in view.

When you irritate the peripheral end of the nerve proceeding to the submaxillary gland—a nerve supplied as we now know from the chorda tympani—the following phenomena are observed. A very abundant secretion of saliva is produced, the quantity may be so large that, in a short space of time, the volume of saliva secreted shall greatly exceed the volume of the gland itself. This fact demonstrates at the outset that we have not to deal here with a simple phenomenon of excretion, or expulsion of previously secreted saliva.

According to the views of Stilling and of Henle, which prevailed at the time Ludwig published his first investigations, one might be tempted to explain the phenomena in question by admitting that the irritated glandular nerve acts upon the veins of the gland, causing them to contract. The augmentation of the tension of the blood, consequent on the venous contraction, would, by this hypothesis, be the cause of the augmentation of the salivary secretion. But Ludwig has shown that ligature of the veins, without concomitant irritation of the glandular nerve, does not increase the

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no wheals perceptible. It is highly interesting to note that when this urtication made its appearance in the loins, the formication disappeared from the upper extremities.

As there was reason to suspect the existence of ocular troubles, we interrogated his memory, and found that he had observed something like a mist before his eyes, especially at night. This disorder had, in fact, reached such a point in January, 1874, that he had given up attempting to read. On the left eyeball, a harmless pterygium was remarked. Applying the ophthalmoscope, Professor Panas found that the fundus of the right eye was normal, whilst there was a very marked excavation of the papilla of the left eye, the fundus of which was slightly congested.

Let us note, in conclusion, that during the continuance of his ailment he complained of great thirst, and of unusual drowsiness after meals."

This patient recovered under treatment by faradization, as related in the treatise already mentioned (S.).

[1] Ludwig, 'Mitth. der Zurich Naturforsch.,' 1851. 'Zeitschr. für rat. Med.,' n. f. Bd. i, p. 255. 'Wiener Med. Wochenschr.,' 1860, x, No. 28, p. 483. See also the works published by Ludwig in co-operation with Becher, Rahn, and Gianuzzi.

secretion of saliva. That second hypothesis should, therefore, also be eliminated.

But perhaps the irritation of the glandular nerve which, as you are aware, has the effect of inducing dilatation of the arteries, may determine the secretion, simply because it momentarily augments the afflux of arterial blood into the gland? This argument is rendered invalid by an experiment made by Ludwig, which shows that, during irritation, the manometric pressure in Wharton's duct is superior to the pressure of the blood in the arterial conduits. Besides, the hypersecretion of saliva from irritation of the chorda tympani is still exhibited, after ligature of the arteries supplying the gland—in the case of an animal killed by bleeding—or even in the case of a head separated from the body. Let us also add this most remarkable fact, namely, the saliva and the venous blood which flow forth from the submaxillary gland, whilst the glandular nerve is being stimulated, present, as MM. Ludwig and Spiess[1] have shown, a higher temperature than the arterial blood which passes into the gland.[2]

Judging from the general bearing of these results, it appears evident that the influence of the nervous system on the submaxillary secretion cannot be explained by the simple phenomena of vascular dilatation and constriction. We are induced to recognize in the glandular nerve a two-fold property, since, in addition to its influence over the vessels, the dilatation of which it determines, it also exerts an immediate action on those parts of the gland which accomplish the chemical act of secretion, or, in other words, upon the secreting cells. This influence of the nerve upon secretion seems, indeed, to be the fundamental fact, for it shows itself, in consequence of excitation, even when the effects of the concomitant dilatation are annihilated. As, on the other hand, it does not appear possible, experimentally, to suppress separately the secretor action, leaving the dilator action alone persisting,[3] it is legitimate to suppose that the latter depends on the former as a more or less direct consequence.

We had, therefore, reason to inquire what might be the link of connection between the excitation of the secretor elements determined by stimulation of the nerve, and the hyperæmia which follows that excitation. Several physiologists have thought that we have here to deal with an *attraction* which the secretor elements of the gland should exert upon the blood. "So that to the force hitherto known as assisting the return of the circulating blood to the heart and which is termed *vis a tergo,* we should add a new attractive force in correlation with the intimate nutrition of the elements, a force named by many authors *vis a fronte.*"[1] Is this a purely theoretical conception, unsupported by experiments, and merely destined to cloak our ignorance? By no means. The works of H. Weber, Schuler, Lister, etc.,[2] contain numerous experimental facts calculated to render evident the *attraction* which the tissues can exercise, under certain conditions, over the circulating blood. I will cite two facts of this sort as examples, in which the phenomena may be studied apart from any intervention of the nervous system. I borrow them from a lecture on the Theory of Secretions, delivered in the Museum of Natural History, by Professor Vulpian.[3]

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[1] Ludwig und Spiess, 'Sitzungsber.,' d. v., Ak. Math. Cl., 1857, Bd. xxv, p. 584.

[2] In reference to this, see a Lecture of M. Vulpian, 'Revue des Cours Scientifiques,' 3d année, 1865–1866, p. 741.

[3] By recent experiments, however, M. Heidenhain seems to have been able to demonstrate that, in the chorda tympani, different nerve-fibrils are devoted to secretion and to circulation in the submaxillary gland. He states that in dogs, placed under the influence of woorari, after injection into the jugular vein of a dose of atropine sufficient to completely paralyze the cardiac filaments of the pneumogastric, the stimulation of the chorda tympani no longer determined the slightest secretion. Nevertheless, there was an acceleration of the venous current which did not notably differ from the acceleration determined by irritation of the chorda, before poisoning. 'Archives de Physiologie,' 4 Juillet, 1872.

If you cut all the nerves of a frog's limb and then determine an excitation by placing a small drop of nitric acid on the skin of the web of its foot, a more or less intense hyperæmia will be produced in this point, at the end of a certain period. The second fact is conclusive. An egg on the fourth day of incubation presents a very distinct vascularity of the umbilical membrane. At that period, there cannot be the slightest question of nervous influence. Now, if you place a small drop of nicotine on any point of this vascular area, there ensues around this point so great a congestion that almost all the blood flows thither. In truth, this hyperæmia, this stasis by irritation of the tissues, displays itself, at first glance, with I know not what semblance of a metaphysical conception. But an effort has long since been made to give an interpretation of this phenomenon on physico-chemical grounds. Thus, in 1844, Dr. Draper[4] remarked that where a capillary tube contains two liquids, of different natures, if one of them have a greater chemical affinity for the parietes of the tube than the other, motion ensues, and the liquid which has the greater affinity pushes the other before it. The arterial blood having a greater affinity for the tissues than the venous blood, saturated with the products of disintegration, it should follow that the venous blood would be driven back. According to this hypothesis, it would suffice to quicken the chemical process of nutrition, in order to increase the intensity of motion (or afflux), and herein the action of the nerves may intervene. The phenomena of stasis are capable of being explained in an analogous manner, by an appeal to the laws of osmosis (blood-stasis, by diffusion).[1]

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[1] Vulpian, 'Revue des Cours Scientifiques,' t. iii, p. 744.

[2] See O. Weber, 'Handbuch der Chirurgerie,' t. i, p. 111.

[3] Vulpian, *loc. cit.*, p. 743.

[4] Draper, "A Treatise on the Forces which Produce," &c., New York, 1844. Savory, 'British and Foreign Review,' t. xvi, 1855, p. 19.

However it be, whatever may be the explanation of the phenomena, you perceive that the attraction which the tissues, under the influence of certain agents, exercise upon the blood is a fact experimentally established, wholly apart from any action of the nervous system. Now, in order to apply this datum to the case of the submaxillary gland, it suffices to admit that the glandular nerve, when subjected to excitation, induces a modification of the intimate nutrition of the secretor cells—and then, in consequence of this change, vascular dilatation would take place.

Anatomy seems, besides, to throw a new light upon the question by showing that the terminations of the glandular nerves penetrate into the secretor cells.[2] Herr Heidenhain has even endeavoured to demonstrate that a gland, of which the nerves have been subjected to a somewhat prolonged irritation, presents a histological constitution differing in some respects from that of a gland in a state of repose. The old cells, termed mucous cells, appear in fact, after the irritation, to be replaced by young cells of recent formation.[3] If the views of Herr Heidenhain be confirmed, we should attribute to the nerve a direct influence, so to speak, upon the development of gland-cells.[4]

The hypothesis which has just been formulated in relation to secretor nerves, might apparently be extended to other nerves in which experimental physiology has discerned the property of determining the dilatation of vessels under the influence of stimuli. These nerves would act primarily on the inter-vascular elements and quicken therein the movements of composition and decomposition. Vascular dilatation would follow, as a consecutive phenomenon. In support of this view, one may here also invoke the teachings of anatomy which, in these latter days, has, it is stated, succeeded in following, at least in the frog, the nerve-endings even into the nucleoli of the corpuscles of the cornea, and of the conjunctival cells of the nictitating membrane.[5]

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[1] O. Weber, *loc. cit.*

[2] E. F. W. Plüger, "Das Nervengewebe der Speicheldrüse," in Stricker's Handbuch, t. i, p. 313.

[3] Heidenhain, "Studien der Physiologischen Instituts," 3e Breslau, 1868, and Stricker's Handbuch, *loc. cit.*, p. 330.

[4] According to M. Ranvier ('Traduction de Frey,' p. 437), and M. Ewald ('Jahresber.,' t. i, 1870–1871, p. 55), the results obtained by Herr Heidenhain ought to be interpreted as follows: Under the influence of the irritation of the gland-nerves, the cells called mucous cells simply lose the mucus they contain and resume the appearence of parietal gland-cells. There would consequently be no formation of new cells here, as Heidenhain asserts.

[5] See Kühne, in 'Gaz. Hebdom.,' t. ix, No. 15, 1862. Lipmann, "Endigung der Nerven im eigentlichen Gewebe und im hinteren Epithel der Hornhaut des Frosches," in Virchow's 'Archiv,' 38e Bd., p. 118, 1869. Eberth, in 'Archiv für Mikros. Anat.,' Bd. iii.

This interpretation was long since proposed by M. Brown-Séquard,[1] and Professor Schiff seems to countenance it when he acknowledges that "active dilatation appears to be alien to the proper coats of the vessels and to take place through the medium of the inter-vascular tissues."[2]

The incursion which we have made into the domain of physiology was undertaken with the intention of collecting, as we went, evidences which we can now profitably apply. It is requisite, indeed, to fix your attention for a while upon the *trophic nerve theory*, as it is called, which, in default of other hypotheses whose insufficiency was admitted, has been sometimes recurred to in order to explain the production of nutritive lesions developed by an influence of the nervous system. Now, by this theory, at least as it has been formulated by Herr Samuel, the supposititious nerves would be, as it were, constructed after the model of the secretor nerves in this respect that, like them, they would exercise, in the normal state, a direct influence over the nutrition of the parts where it is supposed their ultimate terminations are distributed. Their physiological role would be, not to operate directly, but to quicken, throughout the tissues, those exchanges which constitute elementary assimilation and disassimilation, just as the function of the secretor nerves is to set at work in the gland-cell an inherent property, indefeasibly connected with the phenomena of intimate nutrition. The autonomy of the anatomical elements in accomplishing the nutritive act is therefore not at all overlooked; it is only proposed to consider the trophic nerves as forming, in their totality, a perfecting appliance peculiar to superior organisms.

So much for the physiological aspect of the theory: Now, as regards its application to the interpretation of pathological phenomena, it is easy to conceive that a frequent result of morbid irritation set up in nerves, endowed with such properties, would be to carry disturbance into the intimate nutrition of the innervated parts, and to provoke therein, occasion serving, the consecutive development of the inflammatory process. Suppression of the action of these nerves would, on the contrary, have no other effect than that of lessening the intensity of the nutritive movement, and *circumscribed atrophy* is mentioned as an example of the trophic disorders which may thus supervene.

These are the general features of the theory; as to the details, it was to be anticipated that an hypothesis created by the need of explaining as yet but little known phenomena, insufficiently examined at the period of its publication, was destined to become speedily antiquated. That, in fact, is what has happened. It cannot be admitted to-day, that all the trophic nerves have their central origin in the posterior spinal ganglia, or in the analogous ganglia of the cranial nerves; for the cases are numerous, as you have seen, where a lesion, situated in the central portion of the spinal cord, or even in the encephalon, provokes the manifestation of trophic derangements in the peripheral parts. Henceforth, also, we must take count of facts, unknown when Samuel's book appeared, which place beyond all doubt the influence of lesions of the anterior nerve cells on the development of different kinds of myopathies.

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[1] Brown-Séquard, 'Researches on Epilepsy,' p. 70. 'Central Nervous System,' pp. 148, 172, 174.

[2] Schiff, 'Leçons sur la Digestion,' t. i, p. 256.

I have never shared in the disdain with which the theory, that has just been briefly described, was almost universally met. It has ever seemed to me that, in spite of its imperfections, it was worthy of being recommended to the attention of physicians because it explains better, in my opinion, the phenomena which they are called on to observe, in practice, than all the other hypotheses previously invoked. I am very far, however, from wishing to ignore the objections alleged against it. In the first place, the existence of trophic nerves is not, certainly, demonstrated anatomically; it must be admitted, moreover, that most of the experiments made on animals by Herr Samuel, with the object of revealing their existence, have not been felicitous. Some, when repeated by other observers, have not hitherto reproduced the stated results; others have had to be given up, as tainted with numerous causes of error.[1] But all the arguments directed against this theory have not so much value as these. If, for instance, we were bound to condemn the hypothesis of trophic nerves, by the mere fact that it is useless in physiology, I would point out that the utility of the secretor nerves was only recognized, as an afterthought. We should, in like manner, be necessarily compelled to recognize the utility of trophic nerves, if experiments should at any time declare in their favour. Again, it is difficult to believe that the part played by the secretor nerves is absolutely special, and wholly unexampled in the organism. With these nerves we can already compare the dilator nerves, if it be true that they act according to the mechanism already indicated. We should place also beside them, following the recent observations of Herr Goltz, the nerves of absorption which, according to this physiologist, act upon the endothelial cells of the blood-vessels in the same way as the nerves of secretion act upon the glandular epithelium. On the whole, we do not see that any reason exists to decree, *a priori*, that the trophic nerves shall not, some day, be called to a place in this group.[2]

However this be, before adopting a theory which cannot subsist without calling out a whole system of nerves whose existence is as yet problematical, it is necessary to make sure, by every means, that it is really impossible to explain the phenomena, the interpretation of which is required, by appealing to the properties of the different nerves already known. We must take care not to infringe the axiom of Logic, *Haud multiplicanda entia absque necessitate.* Now, the vaso-motor theory being eliminated, there yet undoubtedly remains much to be done from this point of view.

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[1] See Tobias, 'Virchow's Archiv,' bd. xxiv, p. 579, and O. Weber in 'Centralblatt,' 1864, p. 145.

[2] Goltz in 'Pflüger's Archiv,' t. i, v, p. 53, and 'Journal of Anatomy and Physiology,' 2d series, May, 1872, p. 480.

There is one opinion, amongst others, which has not received attention, so far as I know, and which perhaps deserves to be taken into consideration. The numerous and decisive experiments which have been recently made on the connections formed by uniting ends of divided nerves possessing different functions, such as the hypoglossal and lingual nerves for example,[1] have shown that excitations, produced on any point of a sensitive or motor nerve-fibre, are propagated at once and simultaneously in centripetal and centrifugal directions. From this, it is allowable to suppose that pathological irritations, developed on a sensitive nerve, either at its central origin or on some point of its course, reverberating in a centrifugal direction to the ultimate extremities of the nerve filaments, *i. e.*, to the papillæ of the derm, or the substance of the rete mucosum,[2] could there provoke inflammatory action, in certain cases. In this way we could comprehend, for instance, the somewhat frequent development of pemphigoid or bullar eruptions, and of zona, in consequence of lesions affecting the posterior fasciculi of the cord, or the sensitive spinal roots. With respect to the motor nerves, I do not see any serious reason to prevent us from admitting that pathological irritations, affecting the nerve-cells of the anterior cornua, would sometimes be transmitted to the muscular fasciculi, by means of the nerve filaments which, in the physiological state, transmit voluntary excitations. A certain number, at least, of the trophic disorders, consecutive on lesions of the nervous system, would perhaps find their explanation in this hypothesis, without its being necessary to have recourse to the trophic nerve theory.

We have arrived, gentlemen, at the conclusion of this pathogenic discussion, and, as I allowed you to perceive from the beginning, the question in dispute still awaits its solution. I shall not regret, however, the course of explanation we have followed, if, by placing before your eyes the documentary evidences of the case, I have succeeded in inspiring you with the desire of entering more deeply into an investigation which concerns, to such a supreme extent, the pathology of the whole nervous system.

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[1] Vulpian, 'Physiologie du Système Nerveux,' p. 290.

[2] See Langerhans, 'Virchow's Archiv,' Bd. 44, and A. Biesadecki, Stricker's Handbuch, p. 595.

PART SECOND.

PARALYSIS AGITANS AND DISSEMINATED SCLEROSIS.

LECTURE V.

ON PARALYSIS AGITANS.

SUMMARY.—Of tremor in general. Its varieties. Intermittent tremor. Continuous tremor. Influence of sleep, rest, and voluntary motion. Distinction established by Van Swieten. Opinion of M. Gubler. Tremor, according to Galen. Paralysis agitans, and disseminated sclerosis—independent diseases. Parkinson's researches. French works: MM. Sée, Trousseau, Charcot, and Vulpian. Paralysis agitans admitted to the right of domicile in classic treatises.

Fundamental characters of paralysis agitans. A disease of adult life. Its symptoms. Modifications observed in the gait. Tendency to propulsion and retropulsion. Invasion: its modes, slow or abrupt. Period of stationary intensity. Head and neck not affected by tremor. Alterations of speech. Rigidity of the muscles. Attitude of the body and limbs. Deformation of the hands and feet. Delay in the execution of movements. Perversions of sensibility. Cramps; general sensation of tension and fatigue: need of frequent change of position. Habitual feeling of excessive heat. Temperature in paralysis agitans. Influence of the kind of convulsions—static or dynamic.

Terminal period. Confinement to bed. Disorders of nutrition. Enfeeblement of the intellect. Sacral eschars. Terminal complaints: they differ from those of disseminated sclerosis. Duration of paralysis agitans.

Necroscopical results. Inconstant lesions in paralysis agitans; fixed lesions in disseminated sclerosis. Lesions of the pons Varolii and of the medulla oblongata (Parkinson, Oppolzer). Pathological physiology.

Etiology. External causes; violent moral emotions; influence of damp cold, when much prolonged; irritation of certain peripheral nerves. Predisposing causes. Influence of age. Paralysis agitans appears at a more advanced period of life than disseminated sclerosis. Sex. Hereditary predisposition. Influence of race.

GENTLEMEN: Those amongst you, who, this morning, passed through our wards, were probably surprised to find collected there so great a number of female patients, in whom tremor seems to constitute the paramount or at least the most striking symptom of the disease which they labor under. This gathering of patients, forming a genus apart, I purposely contrived. In that way, I desired to enable you to recognize, by means of a comparative study, certain shades of distinction and even marked differences which the examination of isolated cases does not allow you so readily to discern.

At first glance, you may have thought the scene monotonous in character. Indeed, to a superficial observer the phenomenon of tremor in all these women must appear identical, or almost so; one thing alone strikes the gazer's glance, as peculiarly noticeable, it is the diversity in position and intensity of the rhythmical oscillations of the limbs. But a more attentive inspection soon allowed you to distinguish, under this apparent uniformity, different features which at first had completely escaped you.

Thus, to mention merely the most manifest fact, you have been enabled to remark that some of our patients do not tremble except when executing a coördinated movement with a member, as in the act of raising a glass of water to the lips to drink, or again when they attempt to rise from their chairs in order to walk about. In the latter case, every part of the body may be shaken by energetic convulsive movements, rendering it difficult and sometimes even impossible to stand upright or to walk along the floor. On the other hand, when they are at rest and not affected by any strong emotion, these same women, whether seated or lying, present the most natural attitudes; the different parts of their bodies are nowise agitated, and if you saw them only under such circumstances you would certainly not suspect the disease which has possession of them.

In a second series of cases, on the contrary, the tremor is continuous, permanent; it agitates the members unceasingly, allowing them no peace, and if purposed movements exaggerate it at times, repose does not cause its disappearance. In reality, during waking hours, when the affection is intense, there is no truce for these patients. Whatever be the position they assume, whether they sit or lie down, they are always trembling. Sleep alone puts a temporary stop to the spasmodic agitation of their members; but hardly have they awaked than the tremor makes its appearance anew and soon resumes its former intensity.

Thus, if we take count merely of this primary distinction, founded on the influence of repose and of purposed movements over the production of tremor, it becomes already possible, as you perceive, to gather into two principal groups the patients whose cases occupy our attention.

The first group would comprise those in whom tremor is only shown when an intentional movement is made; whilst the patients, in whom tremor is a constant symptom, or from whom it rarely departs, except during sleep, would constitute the second group. It must be noted, however, that each of these groups, far from forming a homogeneous whole, embraces numerous morbid species, of very different natures, in spite of the analogy impressed upon them by the possession of this symptom in common.

The distinction which I strive to make manifest to you is, in my opinion, of the highest importance in the history of chronic diseases of the nervous system, accompanied by tremor. In our own days, it has been almost universally misunderstood, and, if I mistake not you will seek in vain for a trace of it in our classic authors. However, as M. Guéneau de Mussy has justly pointed out in a recent clinical lecture,[1] the physicians of the last century had taken it into consideration, and perfectly understood its value.

Van Swieten, among others, expressly recognized the two kinds of tremor; nay more, he endeavored to connect each of them with a particular physiological condition. Allow me, here, to invite your attention to the commentary on the 625th Aphorism, where you will find a physiological interpretation of the tremor-symptom that is far from being devoid of interest even for a modern reader.

Thus, according to Van Swieten, the tremor which persists, during repose in bed, results from an irritation that affects the nervous centres in an intermittent and rhythmical manner. This would consequently be a convulsive phenomenon—*tremor coactus.*

On the other hand, the tremor which is exclusively shown, during the execution of voluntary movements, would depend on a defect of stimulus, the result of an insufficiency of the nervous fluid whose function it is to cause contraction of the muscles under the influence of the will. This, therefore, would be a paralytic tremor—*tremor a debilitate.*

An interpretation of the phenomena, which does not radically diverge from the foregoing, was published, a few years ago, by M. Gubler, one of the few modern authors who have upheld the distinction between the two kinds of tremor.[2] M. Gubler notes that, in certain cases, the tremor consists, not in a succession of contrary movements withdrawn from the influence of the will, but in alternate contractions and relaxations of the muscles in action, whether employed to alter the position of a limb, or of the whole body, or to preserve the natural attitude of the various parts. Here the muscular contractions, instead of proceeding, as in the normal state, gradually, imperceptibly, and without shock, take place on the contrary by jerks, and as if by interrupted current, with intervals of rest. This pathological condition, which according to M. Gubler might be designated *muscular astasis,* stands distinctly apart from that condition in which tremor is not determined solely by the contractions required to maintain the corporeal attitude or in obedience to mental command, and taking place by jerks. In the second case, involuntary and purposeless contractions really exist, and are incessantly excited by an internal stimulus.

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[1] Mussy, 'Gazette des Hôpitaux,' 1868.

[2] 'Archives Générales de Médecine,' 5e serie, t. xv, 1860, p. 702.

The classification must, in truth, be a most natural one, for it dates from long before Van Swieten: Galen had established it. He also, in fact, distinguished two kinds of trembling: one, which he designated τρεμος (tremor), is the paralytic shake; the other, which he styled παλμός (palpitation), is the clonic, spasmodic, convulsive agitation[1].

But the physiological aspect of the disease must not detain us any longer, for we could not think of entering, at this moment, into a discussion which would be premature. Let it suffice that we have put prominently forward those characters which can be recognized by the simplest observation, irrespective of any theoretical prepossession. It is because these have not been considered, that the two affections which are to form the object of our first clinical studies—*paralysis agitans* and *disseminated sclerosis*—have remained until to-day, confounded under the same rubric, although they are, in every respect, perfectly independent of each other. Both, indeed, reckon tremor amongst their most important symptoms; but, in the first, the rhythmical oscillations of the limbs are nearly quite permanent, whilst in the second they only supervene on the attempt to execute intended movements. We have just pointed out a distinctive character which already enables us to lay down a broad line of demarcation between the two affections. However that is far, indeed, from being the only one which we have to set before you, as you will hereafter perceive.

Paralysis agitans, which shall first engage our attention and of which I have shown you several well-marked examples, was the first to be inscribed on nosological lists. Its history, however, does not reach far back. The first regular description given of it only dates from 1817; it is due to Dr. Parkinson, who published it in a little work entitled "Essay on the Shaking Palsy." From that period, paralysis agitans has been often mentioned in England and in Germany; but in France it remained almost unheard of until these latter years, for, if I mistake not, it was first described, in an explicit manner, by M. G. Sée in his memoir on Chorea, where it figures among the diseases that might be confounded with St. Vitus's Dance.

In 1859, M. Trousseau in his Lectures on Chorea succinctly tabulated the principal characters of paralysis agitans. Three years later M. Vulpian and I published a study on this subject in the *Gazette Hebdomadaire*.[2] We had but recently been appointed to La Salpêtrière. Desiring to inform ourselves fully as to the nature and characters of this disease, which we were called to observe, on a large scale, we were struck by the insufficiency of the details to be found in existing works. This led us to collect the facts which lay under our observation, and, uniting these with the accounts given by foreign authors, we traced a tolerably complete history of paralysis agitans, considering the period.

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[1] G. V. Van Swieten, 'Commentaria,' t. ii, p. 167. Paris, 1771.

[2] 'Gazette Hebdomadaire,' 1861, pp. 765, 816, et 1862, p. 54.

From that date, this disease obtained the right of domicile in classic works. In the second edition of his lectures, Trousseau devotes a considerable space to its consideration. It figures in the last edition of M. Grisolle's book, and in Reynolds's *Encyclopædia*;[1] but in all these descriptions, and our own does not at all escape this reproach, there is complete confusion between paralysis agitans and disseminated sclerosis. The line of demarcation between these two diseases was for the first time indicated by myself, if I mistake not, in the thesis of M. Ordenstein.[2] It behooves us therefore to establish a parallel between these two affections, by comparing them with each other in the threefold relations of symptoms, causes, and lesions; for that purpose, we shall refer to the afore-mentioned documents and to the numerous observations which we have collected in this hospital. It will be easy for you to discern in the patients whom I have gathered together in these wards, the characters which I am about to insist on.

FUNDAMENTAL CHARACTERS OF PARALYSIS AGITANS.

Paralysis agitans, separated from foreign elements, is, gentlemen, at present a *neurosis*, in this sense that it possesses no proper lesion. In the different accounts that have been published we see incongruous lesions mentioned: some of these belong to disseminated sclerosis; others, by their multiplicity and their variability, yield support to our opinion that, up to the present time, paralysis agitans can lay no claim to any definite material lesion.

It assails persons already advanced in *age*, those especially who have passed their fortieth or fiftieth year. This limit, however, is not absolute, for M. Duchenne (de Boulogne) has told us of a case where the patient was a youth of sixteen. However it be, its natural place is amongst the diseases of the second period of life. But it would be going too far to consider it as being a senile disease.

Frequently the *causes* remain unknown. However, of the etiological data two deserve to be cited: 1st, *damp cold*, such as that arising from a prolonged sojourn in a badly ventilated apartment, or in a low dark dwelling on the ground floor, etc.; 2d, *acute moral emotions.* The latter cause appears to be tolerably common. One of the patients, whom you have visited, was seized under the following circumstances: Her husband, one of the *Garde Municipale*, formed part of the troops who fought against the insurgents in 1832. Having seen her husband's horse return riderless to the barracks, she received a great shock, dreading some disaster. That very day she began to tremble, and the shaking, which at first was localized in the right hand, extending gradually, invaded each of the other members in succession. I shall have occasion to quote numerous examples of the same kind for your information.

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[1] J. Reynolds, 'A System of Medicine,' t. ii, p. 184; art. 'Paralysis Agitans,' by W. R. Sanders.

[2] 'Sur la paralysie agitante et la sclérose en plaques généralisée,' Thèse de Paris, 1868. Cohn, however, had remarked that in two cases of multiple induration of the brain and spinal cord, the tremor was only exhibited after movements which the patient wished to make, but never in a state of repose, nor during sleep, "Ein Beitrag zur Lehre der Paralysis agitans," in 'Wiener Med. Wochenschr.,' Mai, 1860.

The *symptoms* of paralysis agitans are not all of the same value. The most striking symptom consists of a tremor, existing even when the individual reposes, limited at first to one member, then little by little becoming generalized, whilst respecting, however, the head. To this phenomenon is superadded sooner or later an apparent diminution of muscular strength. The movements are slow and seem feeble, although dynamometrical experiments prove that this diminution is not real. This motor impotence appears to be due in part, as we shall see, to the rigidity which prevails in the muscles.

A singular symptom is that which, frequently at an early, but usually at a late period, comes to complicate the situation—the patient loses the faculty of preserving equilibrium whilst walking. In some patients also we notice a tendency to propulsion or to retropulsion: without feeling and giddiness, the patient is, in the first case, propelled forward, and, as it were, compelled to adopt a quick pace; the individual is unable, without extreme difficulty, to stop—being apparently forced to follow a flying centre of gravity.

A peculiar attitude of the body and its members, a fixed look, and immobile features should also be enumerated among the more important symptoms of this disease.

The *march* of paralysis agitans is slow, and progressive. Its *duration* is long—sometimes it has gone on for thirty years. The fatal term supervenes either by the advance of age, or because of intercurrent diseases which may be accidental or occasioned by marasmus, confinement to bed, etc. In the first case, an acute disease, such as pneumonia, occurs: in the second, death takes place from a sort of nervous exhaustion, nutrition degenerates, the patient cannot sleep, eschars are formed and conclude the morbid scene.

Such, gentlemen, are the more general characters of paralysis agitans. But in order that you may the better comprehend their significance, it is proper to enter more deeply into a study of the symptoms and to show how they arise, increase, and are connected with the different stages of the disease. To this end, and in order to give greater clearness to our description, we will establish several periods which we shall describe, in due order.

Let us examine, in the first place, the manner of its invasion. Experience teaches that paralysis agitans sets in sometimes slowly and progressively; sometimes, on the contrary, in an almost abrupt manner.

A. *Slow invasion.*—In the immense majority of cases, the invasion is insidious, the disease first showing itself as slight and benignant. The tremor is circumscribed to the foot, the hand, or the thumb. This symptom, apparently of so trifling a nature, long remains solitary. It presents, however, certain characters which require to be recognized, and on which we shall lay stress. Is the hand attacked? Its several segments are seen to oscillate over each other, stirred by an almost pathognomonic motion. The patient closes the fingers on the thumb as though in the act of spinning wool; at the same moment the wrist is bent by rapid jerks upon the forearm, and the forearm on the arm.

At this stage of the disease the tremor may be merely passing and transitory. It breaks out when least expected, the patient enjoying complete repose of mind and body, and it frequently occurs without his being conscious of it. The act of walking (even where the upper extremities are affected), the act of grasping, lifting, taking a pen, writing, any effort at all of the will, may at this epoch often suffice to suspend the tremor. Later on, it will be no longer so. Moreover, as it augments in intensity and persistence, the tremor invades little by little, and not without observing certain rules in its progress, the parts which have hitherto remained sound. If, for instance, it first affected the right hand, at the end of some months or of some years, the turn of the right foot will come; next the left hand, and after that the left foot will be, successively, assailed.

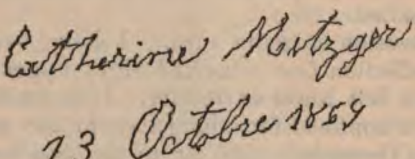
Decussated invasion is more rare. I have, however, at least twice seen the affection first seize the right upper extremity, and pass next to the left lower extremity. It is much more common to see the tremor confined for a long time to the members of one side of the body (*hemiplegic type*), or to the two lower extremities (*paraplegic type*). The head is always nearly quite respected at every stage of the disease, even in the most intense cases; and this is a character to which we shall, hereafter, give prominence, for the contrary is often observed in the cerebro-spinal form of disseminated sclerosis.

I have to ask your utmost attention to a mode of *progressive invasion* which, although exceptional, is not the less worthy of interest. The tremor is not absolutely the first sympton recorded. It may possibly be preceded sometimes by a very remarkable feeling of fatigue, sometimes by rheumatoid or neuralgic pains, which are occasionally most severe, occupying the member or the regions of the members which shall soon be seized, but secondarily, by the convulsive agitation. I might quote several cases of this kind to you, and, in such circumstances, it is not rare to find that some traumatic cause exists, such as a puncture, as Romberg has seen, or, as I have myself observed, a violent contusion of the member which was ultimately taken with pain and trembling. The paralysis agitans which sets in, after this manner, behaves itself, however, throughout its ulterior course in the ordinary way, and its progress is effected in accordance with the same laws.

B. *Abrupt invasion.*—When, in consequence of a moral cause, a shock of terror for instance, tremor suddenly supervenes, it sometimes occupies one member only, sometimes it seizes on all the members at once, from the very commencement. After persisting for a few days it may possibly improve or even vanish. But, later on, after a series of alternate improvements and exacerbations, it takes up its abode in a permanent manner. This, at least, is what we have seen very distinctly occurring in several cases.

The duration of this initial phase varies from about one to two or three years, whatever may have been the manner of its invasion.

C. *Period of stationary intensity.*—When paralysis agitans has acquired its perfect development, the trembling, besides invading several members, becomes, at least in severe cases, almost incessant. Its intensity, however, is not the same at all times. Different circumstances, formerly without influence over it, now augment it. Moral emotions count amongst these, and the exercise of voluntary movements. In addition, we find a kind of crises—paroxysms occurring spontaneously, without appreciable cause. On the other hand, natural sleep, and sleep induced by chloroform, always annihilate the convulsive jerking, for the time being.



Catherine Metzger
13 Octobre 1869

Fig. 7.—Specimen of handwriting in paralysis agitans.

At this stage of the disease, especially, the peculiar characteristics of the tremor are displayed in all their fulness; then also we occasionally find the rhythmical and involuntary oscillations of the different parts of the hand recalling the appearance of certain coördinated movements. Thus, in some patients, the thumb moves over the fingers, as when a pencil or paper-ball is rolled between them; in others, the movements are more complicated and resemble what takes place in crumbling a piece of bread. I have shown you some examples of this kind. These are, if I do not mistake, peculiarities which belong specifically to the tremor of paralysis agitans; I do not believe that they are to be found in any other species. They have been clearly recognized by M. Gubler (*loc. cit.*), who, having occupied the position of clinical clerk in La Salpètrière, was enabled to study the disease on a large scale.[1]

The hands and neck, we repeat, remain unaffected; this is the rule. Far from trembling, the muscles of the face are motionless, there is even a remarkable fixity of look, and the features present a permanent expression of mournfulness, sometimes of stolidness or stupidity. The nystagmus which so frequently figures in the symptomatology of disseminated sclerosis, has no existence in paralysis agitans. Nor are the muscles of the jaws affected by convulsive agitation. Nevertheless, it is not uncommon to find that the tongue, even when inclosed in the buccal cavity, is stirred by a well-marked tremor, which augments when the organ is protruded. Sometimes the lips adhere, as though firmly pressed together, so that the red portion is no longer visible, and the cutaneous surface seems puckered.[2] There is no real difficulty of speech, but the utterance is slow, jerky, and short of phrase: the pronunciation of each word appears to cost a considerable effort of the will. If the tremor of the body be intense it may happen that the utterance will be tremulous, broken, jolted out as it were like that of an inexperienced rider on horseback, when the animal is trotting. However, in both cases, we should only recognize in this a phenomenon of transmitted tremor.[1] Finally, the patients seem to speak between their teeth. Deglutition is accomplished with ease, though perhaps slowly; frequently in cases of somewhat old standing the saliva, accumulated in the mouth, is involuntarily allowed to escape. The respiratory muscles do not seem to share in the convulsive disorder of the extremities. We have to remark, however, that some patients experience an almost continual feeling of oppression.

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[1] The tremor causes the handwriting to exhibit characters which are somewhat special. When the disease is commencing, the writing at first glance seems normal, but when examined with a magnifying glass inequalities are perceived, some parts being thicker and heavier than others. Later on, in the period of stationary intensity for instance, the changes are more marked and consequently plainer. The specimen, Fig. 7 (p. 112), represents the writing of a patient whose case we studied at the Hôpital St. Louis, in 1869. The strokes forming the letters are very irregular and sinuous, whilst the irregularities and sinuosities are of a very limited width (Bourneville).

On a careful examination of this specimen of writing it will be perceived that the down-strokes are all, with the exception of the first letter, made with comparative firmness and are, in fact, nearly normal—the finer up-strokes, on the contrary, are all tremulous in appearance, and it is to the unsteadiness of these lines that the peculiar character of the writing here is principally due. From this I would infer, that the flexors of the fingers were, at this stage of the disease, less affected by tremor than the extensors, and that, amongst the latter, the interossei were probably the earliest affected. This inference will not be without interest for those who remember the attention which Dr. Duchenne (de Boulogne) gave to the conduct of these muscles in the various paralytic and atrophic affections of the hand (Sigerson).

[2] All these characteristics are found well marked in the case of the patient, Marie-Anne Perd. . ., who is still under M. Charcot's care (Salle St. Alexandre, No. 9). The head, held in a somewhat fixed position, is slightly bent forward. The features are, so to speak, without expression; the brow-wrinkles, similar on both sides, are deeply marked; the eyelids are less mobile than in the normal state, on account of a species of contraction in the supraciliary muscle which appears habitual and which deepens the brow-wrinkles. When the patient is requested to close her eyes, she does so, without effort, she says; but the upper lids are then stirred by little convulsive movements which would rather lead one to suppose that it required a certain force to keep them closed. And, in fact, if they be kept in this position, the convulsive movements (a kind of rapid winking) augment in direct ratio with the continuance of the test, so that the occlusion ceases to be complete. The eyes look straight forward: there is no nystagmus. When, in examining the sensitiveness to light of the pupils, we attempt to open and close the eyelids, in alternation, a certain resistance is met with, in the latter

We shall now point out a characteristic which, we believe, was overlooked by Parkinson as well as by most of his successors: we allude to the rigidity to be found, at a certain stage of the disease, in the muscles of the extremities, of the body, and, for the most part, in those of the neck also. When this symptom declares itself, the patients complain of cramps, followed by stiffness, which, at first transient, is afterwards more or less lasting, and is subject to exacerbations. Generally the flexor muscles are the first, as they are always the most intensely, affected. When this muscular stiffness has become permanent it causes the patients, in many cases, to assume a characteristic attitude. Thus on account of the rigidity of the anterior muscles of the neck, the head, as Parkinson remarked, is greatly bent forward, and, as one might say, fixed in that position; for the patient cannot, without much effort, raise it up, or turn it to the right or left. The body also is almost always slightly inclined forward, when the patient is standing.

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case, which is due to the convulsive movements of the upper lids that the patient cannot control. The gaze is, in some sort without expression.

The lips are drawn together and somewhat pouting, as though a muscular contraction maintained them in apposition; it follows that the naso-labial furrows are shallow, like the jugo-mental. The upper lip is motionless, the under lip is moved by a very fine tremor at the labial commissures. The patient is obliged to make an effort in order to open her mouth; she can only open it imperfectly, and cannot keep it open for a few minutes. She appears to account for this customary and as it were permanent apposition of her lips, when she says, "Elles se collent ensemble mes lèvres" (Bourneville).

[1] In reference to *utterance*, we may quote a fragment of the record of the patient, Perd. . . . Utterance began to grow difficult, in this woman, two years ago: and during the last year the difficulty has considerably increased. When the patient speaks, her lips tremble, and the enunciation of the first syllables is laboriously accomplished; her utterance is tremulous especially at first, gradually, as she goes on speaking, the tremulousness grows less, and the words are pronounced in a stronger voice. The patient seems to speak between her teeth; her lips scarcely separate and the jaws are, as it were, adherent each to each. The tongue is stirred by a uniform and general tremulousness, even when lying in the buccal cavity, and when it is protruded the motion augments. The patient asserts that she cannot keep it out long: "It comes back, in spite of me," she says. The mouth is often full of saliva, and Perd. . . attributes to this fact part of her difficulty of utterance (B).

The attitude of the upper extremities deserves to be noticed. The elbows are habitually held a little apart from the chest, the forearms being slightly flexed upon the arms; the hands, flexed upon the forearms, rest upon the stomach.[1] In the course of time, the hands, on account of the permanent rigidity of certain muscles, present deformations which it is necessary to be acquainted with, as they have frequently rendered diagnosis difficult.

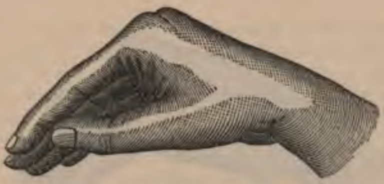

FIG 8.—The writing hand. Habitual attitude of the hand at a somewhat advanced stage of paralysis agitans.

Commonly, the thumb and index are extended and apposed, as if to hold a pen; the fingers slightly inclined towards the palm, are all deviated outwards to the near side (Fig. 8).

The fingers, in many cases, alternately flexed and extended at their several articulations, present a series of inclinations which have a deceptive resemblance to certain types of deformation observed in chronic progressive rheumatism (Figs. 9 and 10).

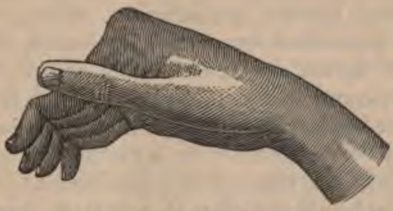

FIG. 9.—Digital deformation, simulating that of primitive chronic articular rheumatism.

The distinction, however, is usually made with ease, if the observer be forewarned and on his guard. In cases of paralysis agitans there is found, in fact, neither the articular tumefaction and stiffness, nor the osseous deposits and cracking sounds observed in nodose rheumatism.

With respect to the lower extremities, the rigidity of the muscles is sometimes so intense as to suggest the existence of real paraplegia, with muscular contracture. In the case of the two female patients, to whom I directed your attention in the wards, their lower limbs, as you have seen, are rigid in semi-flexion; they can neither be flexed nor extended without considerable difficulty. The knees are drawn together in adduction: the feet are stiff, extended, and turned in, simulating the malformation known as *talipes equinus* (*varus*) *club-foot;* the toes are raised and recurved so as to form a *griffe* (or claw) on account of the extension of the first and concomitant flexion of the second phalanges. Nevertheless, these women still retain the power of moving their lower extremities, though slowly indeed and with difficulty: they are even able, as you have witnessed, to walk in a kind of a way, without assistance or support. I have pointed out to you, gentlemen, that, contrary to what takes place in true paraplegia with contracture, we do not here discover that tetanic tremulation, which, whether arising spontaneously or provoked by certain attitudes, characterizes one of the varieties of spinal epilepsy. This symptom, on the other hand, is generally found in the paraplegia which frequently accompanies disseminated sclerosis, and this is a distinguishing character on which we shall have occasion to lay stress in diagnosis.

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[1] See case of the patient Gav. . . described in the appendix. The inclination forward has since become more decided. Moreover, she now exhibits a tendency to incline towards the right side. This lateral inclination is found to exist also in another of M. Charcot's patients named Bau. . .

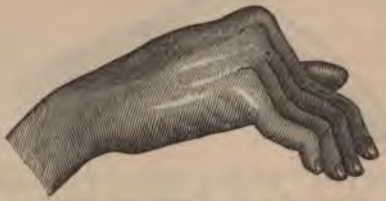

FIG. 10.—Digital deformation, simulating that of primitive chronic articular rheumatism.

As Herr Benedikt has remarked in his recent treatise on Electrotherapeutics, the habitual rigidity of a certain number of muscles undoubtedly contributes, to a great extent, in rendering movement laborious. But this is not, we believe, the only cause that should be recognized. However, it is this rigidity which, determining the general attitude, is the reason that the patients, shrunken upon themselves as it were, seem to move all of a piece; that their joints appear "soldered together," to use a common but fairly descriptive term which I borrow from a patient; and that, finally, the head and body are kept inclined forward—a circumstance which has certainly its own share in producing that tendency to fall forward which the patients experience in walking.

Gentlemen, there are cases, though these are rare indeed, in which muscular rigidity is a symptom of the early stage of the disease, and a really predominant one. I have recently observed an example which belongs to this category. The patient had scarcely noticed the tremor which, in fact, showed little intensity in his case, and was confined to one hand. He already displayed, however, in a high degree, the peculiar attitude of the body and its members, the difficulty of movement, and the characteristic gait.[1] Such cases are exceptional. Most commonly muscular stiffness only appears, or becomes prominent, in the advanced phases of paralysis agitans. Now, when it begins to be manifest, the patients have long experienced, in the execution of movements, a notable want of ease which is due to another cause.

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[1] The following case, which we abridge, belongs to this category of exceptional facts. Guill. . ., aged 53 years (Salle St. Alexandre, No. 10, La Salpêtrière), after having, for some time, suffered from cephalalgia, wandering pains of a lancinating character, and a feeling of constriction at the epigastrium, noticed, four years ago, that the different joints of the right upper extremity were growing stiff. To this phenomenon, weakness was superadded. The stiffness and debility invaded, successively, the left lower extremity, the left arm and afterwards the right leg. In 1870, a tendency to propulsion and retropulsion made its appearance. Thus when the patient ascended the stairs to her lodging, she was propelled forward, and only stopped when she could lay hold on some resisting body; "without this precaution," she said, "I'd upset."

To-day, her condition is as follows: Head slightly bent forward, neck stiff. The brow is deeply furrowed, especially over the eyebrows, which are uplifted as are the upper eyelids: hence the physiognomy has a sort of stolid look. Utterance is free. In walking, the patient takes short steps and keeps the arms close to the body, the forearms flexed, and the hands joined together, as if for support. The fingers, taken together, are slightly bent and gathered; the whole hand is inclined outward towards the ulnar side. All the joints are stiff, but in different degrees, the stiffness being greatest on the right side. Sensation is preserved. During the night, the patient experiences a feeling of cold which passes down from the shoulder to the wrist, and returns in fits lasting from five to six minutes. The members, especially the right arm, feel heavy. When the patient wishes to rise from her chair, and is hindered from helping herself by grasping adjacent objects, she catches the legs of the chair with her hands in order to draw forward the pelvis, then she puts her hands lower down on the sides of the chair, and after some efforts and a kind of balancing to and fro, she succeeds in getting up.

Her slumber is generally short. During the night, Guill. . . keeps herself covered with the sheet only, having a thin petticoat spread over her knees because they are cold. A coverlet, she says, "would be too warm and too heavy." Let us note also an incessant need of change of posture (or fidgetiness). She is scarcely seated for four or five minutes when she asks to be placed further forward, then to one side, &c. Some moments after, she requests that her legs (which have a tendency to adduction) shall be separated; then she begs to be helped to rise. All these symptoms suffice to show that we have here a case of paralysis agitans. Nevertheless, although the disease is of four years' standing, there is scarcely any trembling, the right hand alone being affected by it, and that only for the last three months. From this it will be seen that it is possible to diagnose paralysis agitans, even when tremor is absent (℞.). It was the same with respect to another patient whom M. Charcot observed, a short time ago (1872). This man, aged 50 years, was attacked by "Parkinson's disease" in consequence of a strong emotion occasioned by the attempts of the Federalists, during the time of the Commune, to incorporate him in their battalions. In his case, all the symptoms and especially the attitude were present, but the tremor was likewise deficient. Finally, Mr. Gowers has communicated to M. Charcot a case, noted by him, in the National Hospital for Epilepsy and Paralysis of London; the patient, a woman named Anne Phillips, exhibited all the symptoms of paralysis agitans, excepting the trembling, which is barely perceptible in her movements. (B.) (*Note to the second edition.*)

You will readily discover, in some of the patients whom I have shown you, that laboriousness in the execution of movements which is dependent neither on the existence of tremors, nor on that of muscular rigidity; and a somewhat attentive examination will enable you to recognize the significant fact that, in such cases, there is rather *retardation in the execution of movements than real enfeeblement of the motor powers.* The patient is still able to accomplish most of the motor acts, in spite of the trembling, but goes about performing them with extreme slowness. We noticed this fact a few moments ago, in its relation to the faculty of speech; there is a comparatively considerable lapse of time between the thought and the act. One might suppose that the nervous influx cannot be set to work, in her case, until after extraordinary efforts, and, in reality, the slightest movements occasion extreme fatigue. This group of phenomena has been frequently taken as an indication of a real paralytic enfeeblement. Nevertheless, you will have many an opportunity of assuring yourselves that in cases where the disease has not yet reached its last limits, the muscular power is retained, in a remarkable degree. This fact has been verified, on several occasions, by means of the dynamometer: in some cases, even, the curious phenomenon was noted of the greatest amount of measured force being present in that member which was to all appearance the weakest and most tremulous.[1]

Yet a word upon the *gait* peculiar to patients affected by paralysis agitans. You have seen some of our patients get up slowly and laboriously from their seats, hesitate for some seconds to step out, then, once started, go off in spite of themselves at a rapid rate. Several times they threatened to fall heavily forward. Does this irresistible tendency to adopt a running pace depend exclusively on the centre of gravity being displaced forward by the inclination of the head and body? This explanation, which may, perhaps, be admissible in some instances, will not serve for all. There are, in fact, certain patients who, in contradistinction to those described, tend to run backwards when in motion, and to fall backwards, although their bodies are manifestly inclined forward. Besides, propulsion, like retropulsion, is not absolutely connected with the bent attitude of the body, for it is sometimes seen at an early period of the disease, even before there is any inclination of the body at all.[1] In short, these are not constant and necessary phenomena; they are even frequently enough absent, and are to be found in the symptomatic tables of diseases other than paralysis agitans, as in certain cases of cerebral lesion, for example. It is, however, right to mention that, in the latter event, they are often connected with vertigo, whilst in paralysis agitans the movements of propulsion and retropulsion do not supervene in consequence of any feeling of giddiness.

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[1] We have examined the amount of dynamometrical force present, in six of M. Charcot's patients. The following are the results :—

1st. Perd.—Eight explorations: average on the right side, 60; on the left, 42.

2d. Guill.—Nine explorations: average on the right side, 67; on the left, 63.

3d. Berr.—Thirteen explorations; average on the right side, 59.6; on the left, 41.4.

4th. Gav.—Five explorations: average on the right side, 39.6; on the left, 43.4.

5th. Bean.—Five explorations: average on the right side, 65.5; on the left, 42.3.

6th. Dan.—Five explorations: average on the right side, 41.4; on the left, 33.3.

If these figures be compared with the standard average 85, which we obtained by similar explorations in the case of five persons of the same age as our patients, it becomes evident that the dynamometrical force, far from being preserved in paralysis agitans, is, on the contrary, diminished. It is all the more difficult to explain the divergence between the old opinion and our facts, since in two of our patients this diminution is as well marked at the early stage of the disease as at the most advanced. Finally, in three cases, the dynamometrical enfeeblement is greatest on the side where tremor predominates (B.).

The symptoms which I have just reviewed are not, gentlemen, the only phenomena which deserve to arrest your attention.

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[1] These phenomena are very evident in a patient under M. Charcot's charge (Salle St. Alexandre, No. 22). This woman has attained a more advanced stage of the disease, without however being bed-ridden, than the two patients mentioned in the preceding notes. All the symptoms of the disease are met with in her case; but we shall merely select from her history the facts which relate to propulsion and retropulsion. Suppose, when seated, she is directed to arise and walk, what do we observe? She hesitates for a few moments, then bends the body forward, and after swinging herself to and fro, as it were, suddenly rises. When up, she does not set off at once; she seems to require to poise or balance herself first; she appears in some sort uncertain, the body being inclined forward; finally, she decides to start. Slow at the outset, her gait is gradually accelerated, and, after a course of ten yards, she rushes forward at such a rate that if, at a given moment, she did not find some obstacle to lay hold on—a bed, chair, or wall—she would fall suddenly. In this case, therefore, *propulsion* is as manifest as possible.

Retropulsion is sometimes overlooked because, in order that patients should be conscious of possessing it, they must have had some special occasion to walk backwards. M. Charcot employs a very simple method of exhibiting its existence. When the patient is standing, it suffices to pull her, unexpectedly and slightly, by the skirt, in order to make her immediately commence walking backward; the retrograde movement soon becomes very rapid, and would be quickly dangerous if proper precautions were not taken (B.).

The fact of the phenomena of propulsion and retropulsion being both present in the *same* patient furnishes an argument in support of Professor Charcot's proposition that the propulsive tendency is not absolutely connected with the forward attitude. In this case, the woman when stopped in the midst of an onward walk, being caught by the gown and gently pulled back, immediately began to recede, and this without any perceptible change of attitude. As mention has been made of the existence of a stolid look, in such patients, it will not be uninteresting to note, in relation to the question of the influence of disease on the mental faculties, that although this patient was in an advanced stage of the disease and had the characteristic facial signs, her mind appeared still active (in 1874). "Elle va comme une machine," said an observer, on seeing her walk. "Mais oui, comme une machine à vapeur," was her quick response (Sigerson).

Paralysis agitans is not merely one of the saddest of diseases, inasmuch as it deprives the patients of the use of their limbs, and sooner or later reduces them to almost absolute inaction; it is also a cruel affection, because of the unpleasant sensations which the sufferers experience. Usually, indeed (the neuralgic cases which we have already described being excepted), they are not affected by acute pains, but by disagreeable sensations of a special order. They complain of cramps, or rather of a nearly permanent sensation of tension and traction in most of the muscles. There is also a feeling of utter prostration, of fatigue, which comes on especially after the fits of trembling; in short, an indefinable uneasiness, which shows itself in a perpetual desire for change of posture. Seated, the patients every moment feel obliged to get up; standing, after a few steps they require to sit down. This need of change of position is principally exhibited at night in bed by the more infirm, who are incapable of attending on themselves. The nurses charged with their care will tell you: "They must be turned now on the right side, now on the left, now on the back." Half an hour, a quarter of an hour, has scarcely elapsed until they require to be turned again, and if their wish be not immediately gratified they give vent to moans, which sufficiently testify to the intense uneasiness they experience. In spite of these different troubles, the transmission of the cutaneous sensitive impressions is not altered in paralysis agitans; cold, heat, a pinch, the slightest touch, are all perceived, as in the normal state, and with the wonted rapidity.

But there is one very troublesome sensation which the patients experience, and which I have not found mentioned in any description; this is an *habitual sensation of excessive heat*, so that you shall see them in the heart of winter throw off the bedclothes, and in the daytime only retain the lightest garments. All the cases under our charge give evidence in favour of this assertion. It is a peculiarity worth noticing, although no reason can be given for it, that this sensation of heat is especially felt in the epigastrium and the back. Still it may affect the limbs and face also. It is not of uniform intensity at all times. It appears to attain its maximum after the paroxysms of trembling, and it is then frequently accompanied by profuse perspiration, which is sometimes so great as to necessitate a change of linen; but it may also be found in patients who do not thus perspire and who are but little troubled with tremor.

The knowledge of this fact long since led me to inquire whether the central temperature was altered in these patients. Now experience has proved to me that, whatever may have been the intensity of this subjective sensation or of the tremor, the (rectal) temperature remained at the physiological limit (37.5° C. = 99.5° F.).

You will not be surprised, gentlemen, to find that the muscular contractions, even when so energetic and general as are those we note in certain cases of paralysis agitans, do not give rise to an accumulation of heat in the central parts. These muscular contractions are *dynamic*. Now, you are aware that *static* muscular contractions alone, as M. Béclard has pointed out, occasion an elevation of temperature thermometrically appreciable. From this point of view, as we, M. Ch. Bouchard and myself, have endeavoured to establish, in an essay communicated to the Société de Biologie,[1] convulsions may be classed under two heads:—

1st. *Static*, in which tonic contractions predominate; these augment the temperature to a more or less marked extent. To this category tetanus and epilepsy belong.

2d. *Dynamic*, in which clonic convulsions predominate. These do not affect the temperature in a marked manner. Thermometrical explorations, which we have repeated many times in cases of paralysis agitans, and in some cases of chorea, characterized by excessive agitation, seem to us to have placed the latter point beyond all doubt.[2]

In connection with this question it would be interesting to determine whether the urine, in paralysis agitans, presents any important modification in its chemical composition, and, particularly, any augmentation in the proportion of sulphates, inasmuch as, according to Dr. Bence Jones, such changes take place in chorea and delirium tremens—diseases in which there is great muscular expenditure. This is a desideratum which we propose some day to make good.[3]

Gentlemen, the symptoms we have described to you persist, such as we see them, for a less or greater lapse of time; then, sooner or later, there comes a period that heralds the fatal issue, which may be called the *terminal period.* The affection pursuing its course, the difficulty of movement increases, and the patients are obliged to remain, the whole day long, seated on a chair, or are altogether confined to the bed. Then, nutrition suffers, especially the nutrition of the muscular system. There may supervene, as I have twice observed, a genuine fatty wasting of the muscles. At a given moment, the mind becomes clouded and memory is lost. General prostration sets in, the urine and feces are passed unconsciously, and eschars appear upon the sacrum. In such cases, the patients succumb to the mere progress of their disease, by a sort of exhaustion of the nervous system; and it is perfectly true, as several authors have remarked, that at this terminal period the tremor, however intense it was before, is frequently seen to diminish and even to disappear.[1] On a post-mortem examination, it is not common to find any important visceral lesion capable of accounting for the occurrence of death. We do not observe, for instance, the lesions of caseous pneumonia, or of tubercular phthisis, which, as we shall see, usually terminate the existence of women attacked with disseminated sclerosis or with progressive locomotor ataxia.

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[1] "Sur les variations de la témperature centrale qui s'observent dans certaines affections convulsives et sur la distinction qui doit être établie à ce point de vue entre les convulsions toniques et les convulsions cloniques."—'Mémoires de la Société de Biologie,' 1866.

[2] This statement is corroborated by five new cases. Five explorations made in the case of Ber. gave an average temperature of 37.48° C. In the case of Guill, three explorations gave 37.6°. In the case of Dan. three morning explorations gave 37.3° C.; and four evening explorations, 37.8°. In the case of Grav. two morning explorations gave 37°; and four evening explorations 37.6°. In the case of Bau. three morning explorations gave 37.1°; and four evening explorations 37.45°. The pulse in the first case reached 90; in the second, 86; in the third, 84; and in the fifth, 80. The number of respirations, in these cases, was normal (B.).

[3] Researches have since been made, in reference to this subject, by M. P. Reguard in the laboratory of Sorbonne,—two of the patients in M. Charcot's wards being placed under examination. In both cases, the urine contained a nearly normal proportion of urea, but a less than normal proportion of sulphuric acid. The average of fourteen specimens gave 19.50 grammes of *urea*; and 1.25 instead of 2 grammes of *sulphuric acid*. It follows from these analyses that the excretion of sulphates is diminished in paralysis agitans, contrary to the opinion advanced by Dr. Bence Jones when treating of chorea. In the latter affection, indeed, Lehmann and Gruner have always found a diminution of sulphates. Vogel, on his side, arrives at the same results, and he thinks that the contrary conclusions of Dr. Bence Jones must be attributed to the insufficiency of the analytic method employed.—*Note to Second (French) Edition.*

Such, however, is probably not the most usual kind of death in paralysis agitans. The fatal termination, in fact, is frequently owing to an intercurrent disease. Trousseau thrice beheld death supervene in consequence of pneumonia, and I have noticed the same thing myself in several individuals suffering from paralysis agitans. Was this complication due to the habit which such patients have of remaining uncovered, even in the coldest weather, on account of the sensation of interior heat they experience? We are unable to affirm it.

Let us not forget, gentlemen, that paralysis agitans is one of the grave affections of the nervous system whose duration is the longest. It may last for thirty years. The symptoms of the third period may themselves, as I have witnessed, linger on for four or five years.

If I have dwelt thus minutely upon the symptomatological description of paralysis agitans, it is because it constitutes, even at the present hour, nearly the whole of the history of this disease.

The few autopsies which have until now been made on persons supposedly affected by paralysis agitans may be grouped into three classes.

The first class includes the cases in which no perceptible lesion has been met with in spite of the most attentive explorations.

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[1] In the case of a patient under M. Charcot's charge (Latouil—Marie-Françoise) whose clinical history is given, *in extenso*, in the thesis of M. Claveleira, the trembling completely disappeared the second day before her death (De la Paralysie Agitan,' 1872, p. 35).

Several facts of this kind have been placed on record by different authors. For my part, I have noted three well-marked cases of paralysis agitans in which the results of the autopsies were altogether negative. At other times, we find mention made of common-place lesions, particularly of senile cerebral atrophy; now this may exist, as is well known, without the slightest tremor having been ever present.

The second class comprises the observations published by some authors,—Bamberger, Lebert, and Skoda, for instance,—under the head of paralysis agitans, and in which lesions have been found that, in all probability, pertain to disseminated sclerosis. Such are the cases of Bamberger, Lebert, and Skoda. Was paralysis agitans really the disease under consideration, or were not the clinical symptoms rather those of disseminated sclerosis? The latter was certainly the fact, at least as regards Skoda's case. We shall, however, revert to this question, on a future occasion.

Finally, the last group contains the case given by Parkinson and that of Oppolzer. In Parkinson's case, which, by the way, he relates at secondhand, it appears that there was an *augmentation of volume*, with induration *of the pons Varolii*, *of the medulla oblongata*, *and of the cervical portion of the cord*, and that in addition to this the nerves of the tongue and those of the arm were apparently *tendinous*. The latter necroscopical detail, with others unnecessary to mention, seems of a kind to throw doubt upon the value of this case from an anatomo-pathological point of view.

As to the account given by Professor Oppolzer it is scarcely more conclusive in our opinion, in spite of the importance that has been accorded it. On *post-mortem* examination there was also found an induration of the pons Varolii, which, after microscopical scrutiny, was attributed to a hyperplasia—a proliferation of the connective tissue. What are the characters of this hyperplasia? The narrative is silent respecting them. There is no mention, in the original text, of any atrophy of the nervous elements, nor of any signs of fatty degeneration, two lesions given, for what reason I know not, in the version adopted by Trousseau in his clinical lecture.

The foregoing considerations show, gentlemen, that the special lesion of paralysis agitans remains to be discovered.[1]

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[1] Since this lecture was delivered (in 1868), M. Charcot has had occasion to make three new *post-mortem* examinations. The lesions he met with are of two kinds: Those of the first, constant in these three cases, consisted in (*a*) obliteration of the central canal of the spinal cord by proliferation of the epithelial elements which line the ependyma; (*b*) proliferation of the nuclei which surround the ependyma; (*c*) pigmentation of nerve-cells, most marked in Clarke's column chiefly. Of the second kind of lesion, one was peculiar to two of these three cases, and consisted in a multiplication of the amyloid corpuscles—one was found only in a single case; this was a sclerosed patch on the posterior surface of the bulbus rachidicus. In the most marked case (of paralysis agitans) there was no lesion of the protubentia or of the bulbus discovered. (For further details see Joffroy, 'Société de Biologie,' 1871.)

The *pathological physiology* of the disease is scarcely more advanced than its anatomy. Very shortly, I expect I shall have an opportunity of demonstrating the accuracy of this assertion before you. I shall not dwell upon the subject now, as I desire to conclude the clinical history of paralysis agitans by stating what we know with respect to the etiology and therapeutical history of this disease.

Etiology.—A. Among *external* causes, two especially have a right to be mentioned, in a tolerably large number of cases. First in order comes the influence of violent shocks of the nervous system —fright, terror, the sudden communication of bad news, &c. Instances of this kind abound in books, and the facts which we have ourselves collected oblige us to put away all scepticism on this subject.

Of the female patients whom we have interrogated many related how their complaint took its rise in the midst of the political commotions by which our country has been agitated. It may be sufficient to mention the case of the gendarme's wife, which we have already referred to, and that of a woman at present occupying bed No. 2 in the Salle St. Alexandre, who began to tremble after a violent emotion occasioned by the events of December, 1851. Besides the instances which came under our own observation we may mention—1st, a case of M. Hillairet (recorded in our memoir) concerning a father who saw his son killed before his eyes; 2d, another case, published by Oppolzer, relating to a burgher of Vienna, terrified by the bursting of a bomb beside him;[1] and 3d, a case given by Van Swieten, where a man was suddenly roused from sleep by a fearful thunder-clap. It would be easy to multiply examples, but this could add nothing to what we have already told you. What it behooves you to know is the fact that, in all these patients, the trembling followed immediately, or almost immediately, on the occurrence of the cause. But the peculiar nature of the cause, be it known to you, does not impress any special character upon the disease.

Let us note, in the second place, the *influence of prolonged exposure to moist cold* in the production of paralysis agitans, an influence which, according to some authors, should suffice to establish its rheumatic origin. However, one important fact may be alleged against this view, which is that, neither before the development nor during the course of the disease, do we meet with any form of chronic or acute articular rheumatism, some rare cases excepted. We notice, at most, in cases where the influence of cold can be accused, the presence of wandering rheumatic or neuralgic pains. In this connection we may quote the case of a woman to whom your attention was drawn, and whose (elephantine) gait recalls that of the larger pachyderms. This woman, who was engaged in wafer-making, lived for over ten years in a very damp apartment on the ground-floor, and the description she gives of her unhealthy abode leaves no doubt upon the subject. She was, moreover, exposed to frequent chills in carrying on her trade.

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[1] In a work published in 1873 ('Berliner Klin. Wochenschrift,' No. 24, p. 278, etc.), Dr. O. Kohts reports a number of cases of nervous affections, observed at Strasbourg, which the patients attributed to the terror caused by the bombardment of that city. The author, who expatiates complacently on that disastrous event, informs us that the number of bombs showered upon Strasbourg amounted in thirty-one days to 193,722, making, as he calculates, 6249 per day, 269 per hour, or from four to five per minute. Amongst the pathological cases quoted, three appear to relate to paralysis agitans: one is that of a woman, aged fifty years, another that of a woman aged sixty-one years, and the third that of a man aged fifty-six years. (B.)—*Note to the Second French Edition.*

There are cases in which this cause is, in our opinion, far from having played the part assigned to it. Such is that recorded by Romberg relating to a man who, in 1813, was stripped naked by the Cossacks in snowy weather. Is the action of cold to be accused in this case, or the influence of terror?

Finally, we will point out a third cause, which has been silently passed over, by most medical writers, in describing this disease, namely, the *irritation of certain peripheral nerves*, supervening in consequence of a wound or contusion. A case mentioned by Haas, from Door, in 1852, and quoted by Dr. Sanders, appears to belong to this etiological group. It relates to a girl of nineteen, under whose right toe-nail a thorn had penetrated. She immediately complained of acute pain, and soon after was seized with trembling, which, though at first limited to the wounded foot, gradually became generalized. This trembling, it is said, completely disappeared in the course of time. A termination so exceptional affords us reason to doubt whether this was really a case of paralysis agitans.

The wife of one of our provincial brethren, whom I attended, received a severe contusion of the left thigh, owing to a fall from her carriage. After some time there supervened in the injured limb acute pain following the course of the ischiatic nerve, and, shortly afterwards, the extremity was affected by trembling throughout its entire extent. This tremor, which was at first temporary, became permanent later on, and finally invaded the other members.

With this case we may place that of a midwife, who was also taken with paralysis agitans. This patient, who was under my care in La Salpêtrière for many years, had experienced a violent pain, limited to the course of the nerves of the leg and foot. The parts so affected were the first seized with tremor. The pain, which had arisen spontaneously, and which was at times intolerable, resisted the most energetic remedies. It persisted until the death of the patient, on whom, unfortunately, no autopsy could be performed.

B. We have indicated the cases in which the influence of an etiological element may be discerned; but there are others where the most attentive investigations do not lead to any result. Here we are reduced to inquire into the *predisposing influences*, which it now remains for us to review.

In relation to the question of *age*, we should point out that shaking palsy is not, as has been asserted, a senile disease. It is true that it sets in after forty, and consequently at a later period than disseminated sclerosis. This rule, however, is not absolute; some instances may be cited in which the disease showed itself at an early age, at twenty, for example, as in a case which M. Duchenne (de Boulogne) has told us of.[1]

Sex appears not to exercise any pathogenic influence; paralysis agitans is found as commonly in males as in females.

We possess no precise information with respect to the influence of *hereditary* predisposition. Unlike locomotor ataxia in certain circumstances, and progressive muscular atrophy, paralysis agitans is not a family disease. The observations which have produced a contrary impression relate to partial tremors that showed no tendency to become generalized, and which pertain rather to the class of convulsive twitchings (*tics convulsifs*).

There is some reason to believe that the Anglo-Saxon *race* (in England and North America) is the most subject to this disease. The accounts which I have received from physicians of the countries mentioned, my personal experience, and especially the information with which my friend, M. Brown-Séquard, has supplied me, all yield support to this opinion.

But even in those countries shaking palsy is not *very common.* Dr. Sanders, in his statistical table, which comprises England and Wales, and reaches from 1855 to 1863, records 205 cases of death by paralysis agitans—that is, an average of 22 deaths per year (fourteen men and eight women). Finally, this disease figures in the fifth place, side by side with locomotor ataxia, on the etiological list of affections treated at La Salpêtrière.

Treatment.—A few words, in conclusion, gentlemen, upon therapeutical remedies. It is an incontestable fact that paralysis agitans is sometimes cured. Does this cure take place spontaneously, or because of the remedies employed? The latter hypothesis is but little probable, so far as the majority of these fortunate cases are concerned, for the same drugs, to which the credit of effecting the cure in such cases has been given, have completely failed in other cases. Elliotson administered carbonate of iron, and Brown-Séquard chloride of barium—each of them had a success to chronicle, and, along with that, cases in which the results were negative. M. Duchenne (de Boulogne) has likewise to record the cure of one of his patients. These instances prove that paralysis agitans is not incurable. But we must confess that we are ignorant of the means employed by nature to produce this result.

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[1] M. Fioupe has published in the 'Journal de Médecine et de Chirurgie Pratiques' (p. 389, 1874) the case of a young girl, under the care of Dr. Siredey, who was stricken with shaking palsy at the age of from fifteen to sixteen years: "Towards the end of the siege of Paris she had one day taken refuge in a cellar to be out of the way of the projectiles, when a shell burst, destroying three or four persons at her side. Seized with violent terror, she fainted away, and when, after a few instants, she came to her senses, it was soon observed that her right arm was shaken by a slight tremor, which in a little time invaded the right lower extremity also." She presents, at present, all the symptoms which denote paralysis agitans: characteristic physiognomy, fixity of gaze, special attitude of head and body, peculiar gait, propulsion, retropulsion, &c. (B.)—*Note to the Second French Edition.*

Everything, or almost everything, has been tried against this disease. Among the medicinal substances that have been extolled, and which I have administered without any beneficial effect, I need only enumerate a few. Strychnine praised by Trousseau ('Journal de Beau'), appears to me rather to exasperate the trembling than to calm it. Ergot of rye and belladonna, recommended on account of their anti-convulsive qualities, have not yielded any very profitable results. The same verdict must be given in reference to opium, which, on the contrary, augments reflex excitability, and which was supposed capable of moderating the tremor because of diminishing the pain. Latterly I have made use of hyoscyamine, from which some patients have obtained relief; its action, however, is simply palliative.

Ogle gave Calabar bean without any advantage. As to nitrate of silver, it has always appeared to us to exaggerate the convulsive condition, and this is the more remarkable, because in disseminated sclerosis it sometimes produces a fairly marked amendment, and diminishes the intensity of the shaking.[1]

Finally, we should mention the application of electricity, which, according to some physicians, has brought about several cures. Neither statical electricity nor the interrupted current is recommended. These agents, though of benefit, it is said, in chorea are, at least according to Dr. Gull, inefficient as against paralysis agitans. The continued current of a galvanic pile is prescribed. It is not necessary, gentlemen, to remind you to-day that the physiological and therapeutical effects differ remarkably as you make use of one or other of these kinds of currents. However it be, there are at least two cases in which this method of treatment seems to have proved successful. The first pertains to Remak, the second to Dr. Russell Reynolds. It is proper, therefore, when the occasion offers, to have recourse to the continued current.

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[1] M. Eulenberg has recently recommended the hypodermic injection of a solution composed of one part of arseniate of potash to two parts of water ('Berliner Klin. Wochenschr.,' Nov. 1872). This mode of treatment, having been tried by us in M. Charcot's wards, gave no satisfactory results ('Progrès Médical,' 1874, p. 245). We have also prescribed bromide of camphor in the case of two patients in the same ward, whose disease had lasted for several years. In the first weeks there was amendment of some symptoms, but this improvement did not persist. It might, perhaps, be well to recur to this therapeutic agent in less advanced cases. (B.)—*Note to the Second French Edition.*

LECTURE VI.

DISSEMINATED SCLEROSIS. PATHOLOGICAL ANATOMY.

SUMMARY.—History of disseminated sclerosis; French period; German period; New French investigations; Macroscopic morbid anatomy; external aspect of the patches of sclerosis; their distribution in brain, cerebellum, protuberantia, bulbus rachidicus, and spinal cord. Patches of sclerosis on the nerves. Spinal, cephalic or bulbar, and cerebro-spinal forms. Characters of the sclerosed patches; their colour, consistence, etc.

Microscopic anatomy; sketch of the normal histology of the spinal cord; Nerve tubes; Neuroglia, its distribution; Cortical layer of the reticulum. Characters of the neuroglia, influence of chromic acid. Arterial capillaries. Histological characters of the sclerosed patches; transverse sections; peripheral zone; transition zone; central region. Longitudinal sections. Alterations of the bloodvessels. Examination of the sclerosed patches in the fresh state. Histological lesions consecutive on section of the nerves. Fatty granulations in sections of the sclerosed patches observed in the fresh state. Modifications of the nerve-cells. Mode of succession of the lesions.

GENTLEMEN: At our last conference I dwelt upon the distinction which should be made between the different kinds of tremor. I mentioned, at the outset, that they could be divided into two groups; one, in which tremor is in some sort permanent; another, in which tremor only supervenes on purposed movements. Then, proceeding from these notions, I cited as an example of the tremor characteristic of the first class, that observed in *paralysis agitans*, the history of which I traced for you. On our way, I noted some of the characters which enable us, in these days, to distinguish this disease from another affection, previously confounded with it, namely, from *disseminated sclerosis.*

To this affection, which offers us an example of tremulation belonging to the second class, *i. e.*, a tremor which only appears under certain conditions, we shall devote the present and succeeding lectures. Anatomically considered, disseminated sclerosis forms a clearly defined pathological species; clinically, the case is different, and in this connection we shall have many blanks to fill up. Let us begin by a few words on the history of the subject.

HISTORICAL NOTE.

Disseminated sclerosis is found mentioned, for the first time, in Cruveilhier's 'Atlas d'Anatomie Pathologique,' 1835–1842, an admirable work, which ought to be more frequently consulted by all who desire to avoid the disappointment of making second-hand "discoveries" in morbid anatomy. In Parts 22 and 23 you will observe representations of the lesions found in disseminated sclerosis, and, side by side, you can read the clinical observations which relate to them. I take advantage of this opportunity to commend to your perusal a remarkable chapter on paraplegia. Previous to this epoch, so far as I am aware, there is no trace of disseminated sclerosis to be discovered anywhere.

After Cruveilhier, Carswell in the article on "Atrophy," contained in his 'Atlas,' 1838, has had lesions depicted which pertain to disseminated sclerosis. But this author, who has drawn the materials of his work chiefly from the hospital of Paris, does not relate any clinical case in connection with this subject. Even today I do not believe that disseminated sclerosis is known in England.[1] I do not find it indicated in any of the standard works published in that country, not even in Dr. Gull's valuable collection.[2]

Thus, up till that time, the principal documents in connection with this disease had been collected in France. From that period forth, during a lapse of several years, this question dropped into almost complete oblivion, and we have to seek in Germany for new indicia. Ludwig Turck published, in 1855, examples of lesions manifestly belonging to disseminated sclerosis; still the physiological aspect of it alone engaged his mind;[3] Rokitansky indicates them in his treatise;[4] Frerichs[5] and Valentine[6] record two observations; Rindfleisch,[7] Leyden,[8] and Zenker,[9] present in their turn some elements towards the solution of the problem. There were, however, desiderata to be supplied, and new researches were indispensable. It was at La Salpêtrière that the question of disseminated sclerosis once more attracted attention amongst us. In 1862, M. Vulpian and myself placed new examples on record. M. Bouchard, founding his remarks on the cases collected by us at the Salpêtrière, reopened the subject in a treatise which he read before the Medical Congress at Lyons.

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[1] This lecture was delivered in 1868.

[2] Cases of Paraplegia in 'Guy's Hospital Reports,' 1856–1858.

[3] "Beobachtungen über das Leitungsvermogen des Menschliden Rüchen marks," 'Sitzungsberichte der Kais. Akademie der Wissenschaften, Math. Natur. Class.,' t. xvi, 1855, p. 229.

[4] 'Lehrbuch der Pathologischen Anatomie,' 1856, Zweiter Band, p. 488.

[5] 'Haeser's Archiv,' Band x.

[6] "Ueber die Sclerose der Gehirns und Rückenmarks" ('Deutsche Klinik,' 1856, No. 14).

[7] "Histologische Detail zu der Grauen Degeneration von Hirn und Rückenmarks" ('Virchow's Archiv,' B. xxvi, Heft und 6, p. 474).

[8] "Ueber graue Degeneration des Rückenmarks" ('Deutsche Klinik,' No. 13, 1867).

[9] "Ein Beitrag zur Sclerose des Hirns und Rückenmarks" ('Zeitschrift für Rat. Medizin,' B. xxiv, Heft 2 und 3).

In the preceding enumeration of authorities, we have taken count chiefly of the works relating to the pathological anatomy of the subject, as we propose to make mention hereafter of those which contain clinical details. To the items of information culled from the writers whose names are mentioned above, we shall add other information drawn from unpublished sources, and, in order to facilitate the understanding of our studies, we shall place before your eyes the anatomical preparations which we have preserved.

MACROSCOPIC ANATOMY.

Disseminated sclerosis, as I have informed you, gentlemen, is not an exclusively spinal affection. It invades the cerebrum, the pons Varolii, the cerebellum, the bulbus rachidicus, as well as the spinal cord. We shall, therefore, enumerate the alterations which are to be observed, taking the most distinctive cases, in the different parts of the nervous system, viewing them first externally, then in section.

We have here to consider a comparatively coarse alteration, and it is surprising that it should have escaped observation so long. On the plates before you, in which these changes are accurately depicted, you see the spinal cord spotted with grayish patches, having a more or less regular outline, but in every case distinctly circumscribed and contrasting widely with the adjacent portion of the cord.

Sometimes discrete, sometimes confluent, these spots or patches, as you may easily perceive, are disseminated without any apparent order, and as it were by chance, over the whole of the spinal cord. The medulla oblongata itself does not escape, far from it; different portions of the encephalon are also frequently affected.

But we cannot content ourselves with this simple sketch, and we must enter upon the details of a more regular description. At the outset, we should remark that a mere *external examination* will give but a very imperfect idea of the lesion. The spots or patches which we have mentioned are not superficial; they constitute real kernels or foci which penetrate into the substance of the tissues. Frequently, indeed, the section itself will reveal the existence of other spots concealed in the interior.

Let us first examine the *encephalon.* The general appearance of the cerebrum proper has undergone no modification of form, nor, we may add, of colour; for the patches are very rarely found on the gray substance of the convolutions. It is different, however, as regards the central parts. There, in fact, we find the patches, especially on the walls of the ventricles, in the white substance of the centrum ovale, the septum lucidum, the corpus callosum, and finally in certain regions of the gray matter, *e. g.*, the optic thalami, and the corpora striata.

The *cerebellum* generally presents only internal patches, which occupy especially the corpus rhomboideum.

The *bulbus rachidicus*, the *pons Varolii*, and the different districts of the isthmus, are very frequently affected by patches of sclerosis which, in such cases, are both peripheral and deep seated. When the bulbus rachidicus is attacked, the patches are found to affect, either singly or simultaneously, the corpora olivaria, pyramidalia, restiformia, and the posterior region where the nuclei of origin of the bulbar nerves are disposed. As regards the pons, the patches generally occupy its antero-inferior aspect. If we ascend higher, we see the corpora mammilaria (or albicantia) and the crura cerebri affected.

We now come to the *spinal cord.* Through the pia mater we often perceive the gray spots which assume a rosy tint or salmon-colour, on contact with the atmosphere. But it is especially after the removal of this membrane, a removal easily effected, that the lesions are clearly perceived. They affect the whole of the cord, being found in the cervical, dorsal, and lumbar regions; they invade all the different columns indiscriminately, are to be found on the sides of the fissures, and attack the gray substance as well as the white.

The *nerves* themselves do not escape sclerosis. We see them, indeed, sometimes emerge from a sclerosed patch and yet remain perfectly sound; at other times, we find them affected, in their course, by sclerosed patches quite similar to those of the nerve-centres; this is so, at least, as regards those parts of the nerves which lie adjacent to the centres, for the observations of MM. Liouville and Vulpian (which have been frequently verified) leave no doubt upon the subject.

The *cranial* nerves that have been found to present sclerosed patches are the optic, the olfactory, and the fifth pair. As to the *rachidian* nerves we only know that such patches have been observed on the posterior and anterior roots; but we are not aware whether they have been thus affected in their extra-spinal course.

I will not dwell any further, gentlemen, on the topography of the patches of sclerosis; still, I cannot refrain from requesting your earnest attention to the interest which belongs to this question.

You observe, in fact, that these patches, in different cases, occupy very different regions of the nervous centres, and it is clear that this variation of position should be represented by very different functional disorders. It is, indeed, to this fact that the disease owes a large portion of its protean character. We shall return to this subject; at present, you will remark that these differences of position give rise to certain important clinical divisions. Sometimes the patches occupy the spinal cord exclusively (*spinal form*); sometimes they predominate in the encephalon (*cephalic* or *bulbar form*); finally, the simultaneous existence of patches in the encephalon and cord supplies the *cerebro spinal form.*

In order to close what I have to say respecting the macroscopic anatomy of the disease, it only remains for me to indicate the principal characters which the patches present when considered in themselves.

Sometimes they are salient, and, as it were, turgescent; at other times they are on a level with the adjacent parts; finally, they are sometimes depressed, when of old date.

Their colour resembles that of the gray matter, from which it is difficult to distinguish them; but on contact with the atmosphere they assume a rosy hue, and numerous vessels are observed distributed through them.

These spots have a firm consistence, and, on section, present a clean surface, whence exudes a transparent fluid.

Such, gentlemen, as regards its simple anatomy, is disseminated sclerosis, or sclerosis generalized in patches; we have now to enter upon its minute histological details.

In order to bring to a successful issue this undertaking, which relates to facts demanding a careful exposition, I must beg you to grant me both your entire attention and all your indulgence.

MICROSCOPIC ANATOMY.

The method to be followed is simple. We should proceed from a consideration of the normal state of the tissues; that once known, it will be more easy to deduce the morbid alterations. The preliminary knowledge of the characteristics of the normal state, as regards the organs and the elements whose changes we wish to study, is doubtless familiar to you, and we might, if necessary, enter at once upon an examination of the intimate lesions. Nevertheless, as you are aware, the histological anatomy of the nervous centres is, in many respects, quite new; many of the questions which it suggests are still disputed; whilst, on the other hand, for the comprehension of pathological lesions, it is not a matter of indifference to have a more or less well-grounded opinion in relation to these questions. These considerations induce us to remind you, at least in a succinct manner, of certain fundamental facts of normal anatomy. We shall, however, occupy ourselves chiefly with the spinal cord, an organ of less complexity and more easily studied than the brain. In order to limit the field of our examination we shall not delay to describe the nerve elements, properly so called, whether tubes or cells, nor shall we dwell on their reciprocal relations or modes of grouping, in the formation of what is known as the white and gray substances. We propose to concentrate your attention on the connective gangue (or matrix) which surrounds these elements on every side. To this connective setting a high interest attaches, especially for the pathologist, because we must attribute to it a supreme part in the causation of certain alterations of the nervous centres, and particularly in the cases which at present engage our attention.[1]

I.

A. It will be, I believe, of advantage to inaugurate this study by an examination of thin transparent sections, taken transversely from segments of the spinal cord, which have been properly hardened in dilute chromic acid and coloured with carmine. Carmine is here a precious reagent. Thanks to it, certain elements which under its influence assume a vivid hue are thereby set in relief, whilst other elements preserve their usual appearance. Thus the ganglionic cells, their nuclei, their neucleoli, and also the prolongations of these cells, are strongly coloured under the influence of this reagent. The connective matrix also becomes tinted throughout its whole extent, but much less vividly; as regards the nerve-tubes, the axis cylinder alone takes the carmine tint, whilst the medullary sheath completely resists its action.

All the details which this mode of preparation brings out may be followed on the plate, copied from Deiters,[2] which I here exhibit; you will afterwards easily find them in the very beautiful sections which I shall have passed before you for inspection, and which I owe to the courtesy of Dr. Lockhart Clarke. These sections should be at first examined with a low magnifying power.

In the preparations, as in the plate, the portions pertaining to the white substance of the cord seem to you at first sight almost entirely composed of small regularly rounded bodies, like disks, placed side by side, and having all nearly the same diameter. These are thin cylindrical segments, resulting from section of the nerve-tubes, which tubes are, in that part of the cord, disposed longitudinally, having the same direction as the greater axis of the organ, and are here, like the prisms of a basaltic causeway, placed parallel each to each. In the centres of these disks, which are chiefly formed of non-tinted medullary matter, having a brilliant and transparent appearance, you will see a point or rather a little globule, *i. e.*, the axis cylinder, coloured carmine.

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[1] It is known that the first studies of the connective matrix of the spinal cord date from 1810, and are due to Keuffel; but it is less known that Cruveilhier in his article on apoplexy in the 'Dictionnaire de Médecine et de Chirurgie pratiques,' published in 1820, mentioned it:—"Le tissu cellulaire séreux extrêmement délié qui unit et sépare les fibres cérébrales et qui forme une trame excessivement tenue" (*loc. cit.*, p. 209).

[2] O. Deiters, 'Untersuch. über Gehirn und Rückenmark,' Braunschwieg, 1865, Pl. iii, fig. 12.

A somewhat more careful scrutiny soon enables you to note that the disks in question are not exactly in contact, and that they are, on the contrary, more or less plainly separated, each from its neighbour, by an apparently homogeneous substance, which the carmine colours faintly, and which seems to fill like a cement all the interstices that the nerve-elements leave between them. This substance is nothing other than the connective gangue (or matrix) as we called it a little while ago, the neuroglia of Virchow, and the reticulum of Kölliker. In studying its mode of distribution and arrangement, in the different parts of the section, you will readily perceive that it constitutes an important portion of the mass of the organ. Observe, in the first place, that in the peripheral part of the section it forms a ring or rather a zone of some thickness, from which the nerve-tubes are altogether absent. This zone is covered externally and enveloped, as it were, by the pia mater, with which it contracts only some frail attachments; it is, besides, perfectly distinct as regards structure from the latter membrane, which is composed of fibrillary connective tissue and is, therefore, built up quite differently from the neuroglia. This zone has been carefully described by Bidder, and by Frommann,[1] who term it the *cortical layer* of the reticulum (*Rindenschlicht*); we shall see, further on, that, considered from a pathological point of view, it is of incontestible interest.

From the internal border of this zone or cortex we see septa arise and proceed, at certain intervals, which direct their course towards the centre of the cord, which they divide into triangular compartments of almost equal size, whose bases are at the circumference, and whose apices are lost in the gray matter. Each of these septa gives off secondary dissepiments, and these tertiary, which are again subdivided. Their ramifications get interwoven, crossing and anastomosing, so as to produce a web or network with meshes of unequal size. Each of the largest of these meshes may inclose a fasciculus of eight or ten nerve-tubes, or even more, whilst each of the smaller meshes usually contains only one. The reticulated character described becomes especially evident in those portions of the preparation where, owing to the peculiar distribution of the nerve-tubes, the connective skeleton persists alone.

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[1] C. Frommann, 'Untersuch. über die normale und patholog. Anatomie des Rückenmarks,' Jena, 1864.

The neuroglia plays, probably, a more important part in the gray substance than in the white; there are, in fact, some regions almost exclusively formed by it, as, for instance, the borders of the central canal and the column of the ependyma. It is also predominant in that part of the posterior cornua known as the gelatinous substance of Rolando; in the posterior commissure, which consequently takes, almost throughout its whole extent, a rosy tint in the preparations coloured with carmine, whilst the anterior commissure, on the contrary, in consequence of the numerous transverse nerve-tubes it contains, is much less affected by the reagent. In the gray substance also, as in the white, the neuroglia presents a reticulated appearance; but, in the former case, the greater intricacy of the trabeculæ causes the meshes to be notably smaller, and the whole to assume the appearance of a spongy tissue. In both states, however, it serves as a framework and support for the bloodvessels.

B. It is incumbent on us now to investigate, by means of more powerful lenses, what is the histological constitution of this connective gangue or web, of which we only know as yet the most superficial appearances. Have we here a common connective tissue (laminous or fibrillary)? Certainly not. All observers are agreed upon this point. But, beyond this purely negative notion, almost everything else is disputed in the histological history of the neuroglia. However, one opinion tends to become prevalent here, and this opinion, if I may judge from impressions arising from personal observations, closely approximates to the truth. According to this view the neuroglia would be formed, like the stroma of lymphatic glands, for instance, after the type of simple reticulated connective tissue (Kölliker); that is to say, it would be essentially composed of stellate cells, generally poor in protoplasm, having slender prolongations, ramified several times, whose branches unite with other, so as to bind into one system the several cells, and to render them, as it were, joint-partners [Kölliker,[1] Max Schultze, Frommann].[2] In this form of the connective tissue there exists but very little amorphous matter in the meshes of the reticulum, and the intermediate fibrillary substance, which is one of the fundamental characteristics of the laminous tissue, is completely deficient here.

Let us now see what direct examination enables us to discern in thin sections of the cord hardened by chromic acid and coloured with carmine. As in the case of the stroma of lymphatic glands, which we have just taken for example, it becomes us here to distinguish, in the first place, the cells, and, in the second place, a network of fibroid trabeculæ, which binds these cells together. Let us first take note of what is observed in the white substance. The points of the reticulum, where several trabeculæ meet, form here and there swellings or nodes of different degrees of thickness, situated at almost equal distances from each other. Now, all these nodes, but especially those remarkable by their great size, present towards their central portion a definite, rounded, and somewhat oval corpuscle, more vividly coloured by carmine than the adjacent parts. These bodies are nuclei, having a well-defined border; they are finely granular, are devoid of nucleoli, and measure each on an average from 0.004 m. to 0.007 m. They are soluble in acetic acid, which causes them to shrink in every direction, and diminishes their diameter sometimes by one half; they are known by the name of *myélocytes* (Ch. Robin),[1] or *neuroglia nuclei* (Virchow).[2] A thin layer of protoplasm, having no distinctly cellular appearance, usually surrounds these nuclei (*myélocytes, variété noyau*), which, at other times on the contrary, are inclosed in a genuine rounded or stellate cell (*myélocytes, variété cellule*), and furnished with more or less numerous prolongations (from three to ten, according to Frommann), of different lengths.[3]

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[1] Kölliker, 'Gewebelehre,' 5e Ed., Leipzig, 1867, § 108.

[2] *Loc. cit.*

The prolongations appear to unite with the trabeculæ of the reticulum, which continue them, as it were, without any perceptible line of demarcation. In cases where the cellular form is not distinct the nuclei, either naked or covered only with a thin layer of protoplasm, look like centres whence arise the trabeculæ of the reticulum, and whence they radiate in different directions.

The trabeculæ should be studied in their turn, and considered independently of the connections they have either with the nuclei or with the cells which occupy the nodes of the reticulum; their texture varies somewhat, according as we examine them in transverse and in longitudinal sections. In the first case they have the appearance of thin homogeneous and brilliant dissepiments of a fibroid nature. Anastomosing, they form meshes, the smallest of which is still large enough to inclose a nerve-tube. Suppose we observe a longitudinal section? The trabeculæ are seen to ramify to an indefinite extent, and produce a network having much finer meshes. This network is disposed in the form of dissepiments, which separate the nerve-tubes from each other and inclose them, like a sheath. The interstices which exist here and there between these sheaths and the nerve-tubes seem to be filled up by a small quantity of finely granular amorphous matter. In the normal state we meet nowhere, amongst these trabeculæ, with the slender fibrillæ which constitute an integral part of laminous tissue.[4]

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[1] Robin, 'Programme du Cours d'Histologie,' 1864, p. 46; 'Dictionnaire Encyclopédique,' 2e Série, t. i, 1re part; art. Lamineux, p. 284.

[2] Virchow, Die Krankhaft, Geschwülste, 1864–5, t. ii, p. 127.

[3] In reference to this subject see Hayem and Magnan, 'Journal de la Physiologie, etc., No. 1, 1876. Hayem, 'Études sur les diverses formes d'Encéphalite,' 1868.

[4] The term *tissu lamineux* was applied in 1799 by Chaussier to the tissue recently

In the gray substance the neuroglia is disposed on the same general plan; the meshes of the fibroid network are, however, and especially in parts where the nerve-elements disappear, more serried than in the white substance, and from this cause a spongy appearance results, which we have already noticed. Let us add that the stellate cells abound in greater number than elsewhere in certain regions of the gray substance, and that they sometimes attain such a development that it becomes very difficult to distinguish them from nerve cells; but we shall have occasion to refer to this point again.

A dense fibroid network, with close meshes and numerous cells, is found also in those parts of the white substance where no nerve-tubes exist, in the cortical layer (*Rindenschlicht*), for example, and in the greater septa which arise from it.

If we can depend upon the preceding description it is incontestable that the neuroglia deserves to be classed with the reticulated connective tissue type, whose essential characters we noted a little while ago.

But this description has been chiefly traced out, as you have not forgotten, from observations made on fragments of spinal cord that have been subjected to the action of chromic acid for a greater or less extent of time. Now, can it be asserted that the results obtained by this method of preparation are beyond the reach of criticism? Such is not the opinion of some authors, amongst whom we must cite, in the foremost place, such masters as Henle and Ch. Robin.[1] According to these, the fibroid reticulum, above described, has no real existence, but is an artificial product.

In the fresh state, before the application of reagents, the spaces between the nerve-tubes are filled (according to them), not by solid trabeculæ, arranged so as to form the meshes of a network, but simply by a soft, grayish, finely granular substance, in the midst of which the myelocytes are, as it were, held in suspension.

This substance has the property of hardening, without loss of volume, under the influence of alcohol and different reagents, of chromic acid in particular; and it is owing to this circumstance that it presents a reticulated appearance in preparations treated by the latter reagent. To these objections, arguments or rather facts have been opposed, some of which possess, we think, almost absolute authority. It is conceded that in the normal state an amorphous matter exists, interposed between the nerve-elements, though in scanty proportion (Kölliker), and that this matter possesses the

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and still commonly known as cellular tissue, which we now call connective tissue. Professor Robin still makes use of Chaussier's term, alleging it to be the best, because he says the ultimate elements of this tissue are long filaments, somewhat flattened, thin, slender, soft, and hyaline, smooth, slightly elastic, fasciculated. (Sigerson.)

[1] 'Dict. Encyclopédique,' *loc. cit.*

properties which have been described. It is also conceded that, in the fresh state, the reticulum is less distinctly defined than in preparations hardened by the use of acids. But it is not the less true that, even in the fresh state, thin sections of the white substance of the cord, when placed in iodized serum and dilacerated under the microscope, permit us clearly to discern on their borders the fibroid tractus of the connective tissue (Kölliker, Frommann, Schultze). This result, which it is easy to arrive at in the normal state, is still more readily obtained in certain pathological conditions when the normal arrangements are exaggerated, without being radically altered (Virchow).

This is what takes place, for instance, as we shall point out, in subacute interstitial myelitis and in sclerosis proper, when the alteration has not as yet exceeded the first phases of its evolution.

From all this it has been concluded, and we think the conclusion legitimate, that the chromic acid has no other specific effect than to bring out into better relief the reticulated texture of the connective gangue or setting of the spinal cord. The arrangement was previously in existence; it is not produced from nothing by the action of the reagent.

In order to conclude the remarks which I have thought it my duty to offer you, in reference to the normal histology of the spinal nerve centre, I have only a word to add concerning an anatomical peculiarity of the smaller vessels, and principally of the arterial capillaries, in the substance of this organ. They possess, like the intra-encephalic arterioles, that supernumerary coat which is commonly known as the lymphatic sheath or Robin's sheath. This sheath is separated, as you are aware, from the adventitious coat by a free space, filled by a transparent fluid in which float some definite elements. You will soon recognize the interest belonging to this anatomical arrangement, when we come to the question of interpreting certain lesions.[1]

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[1] Since this lecture was delivered, several works have been published on the structure of the neuroglia (see in reference to this subject a critical review by Gombault, 'Archives de Physiologie,' 1873, p. 458). In an important work, M. Ranvier, whose labours have contributed so much to our knowledge of the connective tissue, has shown that the cells, described by Golgi and Boll, are probably artificial products, due to the preparatory method adopted. The connective tissue of the nerve centres is but little different in structure from that of other regions (Ranvier, "Sur les éléments conjonctifs de la moelle épinière," in 'Comptes-Rendus de l'Académie des Sciences,' Décembre, 1873). The neuroglia is composed of little connective bundles of from 0.001 mm. to 0.002 mm. in diameter. "They do not anastomose," says M. Ranvier, "but in some points they cross each other to the number of four, five, six, seven, eight, and even more. At this crossing there is often a round or oval nucleus, having little nucleoli, which is flattened and surrounded by a granular zone. With a good immersion object-glass, magnifying from 600 to 800 diameters, it is easy to perceive all these details and to discern in the granular zone a lamina of protoplasm which, with the nucleus, constitutes a little flat cell of the connective tissue. Beneath and above this cell the little fasciculi follow each other. It does not

II.

After these preliminaries, it becomes easy for us, gentlemen, to enter upon the study of the histological alterations of the spinal cord in *disseminated sclerosis*. The description of these changes, which we are about to give, will be based, principally, on the results of the investigations to which M. Vulpian and myself have long devoted our attention. We shall also have special opportunities of using, after due revision, the researches made, previously or since, on the same subject by Valentiner,[1] Rindfleisch,[2] Zenker,[3] and especially by Frommann,[4] who, in reference to the examination of a small fragment of spinal cord, has written a large volume, adorned with remarkable plates, and enriched with valuable documents.

We shall describe in the first place the appearances which may be discerned: 1st, on transverse sections; and 2d, on longitudinal sections of fragments of the cord hardened by chromic acid. Then we shall describe, from the examination of fresh specimens, certain peculiarities which are not observable in hardened sections. In both cases the coloration of the parts, effected by means of the ammoniacal solution of carmine, will be here, as well as in the case of normal specimens, an auxiliary of great utility of which it is proper to avail ourselves.

A. When you examine with the naked eye a segment of spinal cord affected by a patch of sclerosis, the morbid part appears divided from the healthy portion, in an abrupt manner, without transition, by a definite line of demarcation. Now this is an illusion. Microscopical examination, even when a low power is used, enables us to state that the apparently healthy region bordering the sclerosed patch really presents, to a certain width, very plain traces of alteration. When you pass the apparent limit of the sound parts the lesions become more marked, and they augment gradually in intensity as you approach the centre of the patch,

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seem doubtful to me," M. Ranvier adds, "that this grouping has been taken for a ramified cell; but that is an error which will be abandoned, I am sure, by all who accurately follow the same method" (which he indicates). On other points of the same preparation may be seen isolated cells, and again (stellate) crossings without cells; these appearances leave no doubt as to what should be the proper interpretation of the foregoing facts. The reader will be less surprised at the numerous conflicting opinions published as regards the neuroglia, if he will recall the numerous discussions excited by the question of the structure of the connective tissue of the peripheral organs. The real nature of this structure has been only revealed by recent researches. (*Note to the Second Edition.*)

[1] Valentiner, 'Deutsche Klinik,' 1856, p. 149.

[2] Rindfleisch, 'Virchow's Archiv,' 1863, t. xxvi, p. 474.

[3] Zenker, 'Zeitsch. der Ration. Mediz.,' 1865, Bd. xxiii, 3 Reih. p. 226.

[4] Frommann, 2 Theil, Jena, 1867; see also Rokitansky, 'Sitzungsber.,' K. M. Klasse, t. xiii, 1851, p. 136; Charcot, 'Soc. de Biologie,' 1868; Bouchard, 'Soc. Anat.,' 1868; Hayem, 'Études,' etc., *loc cit.*, p. 121.

where they acquire their highest degree of development. Whilst proceeding thus, from the circumference to the centre, we are led to recognize the existence of several concentric zones, which answer to the principal phases of the alteration.[1]

a. In the *peripheral zone* the following appearances are observed: The trabeculæ of the reticulum are markedly thickened; sometimes they have acquired a diameter twice as great as that possessed in the normal state. At the same time, the nuclei which occupy the nodes of the reticulum have become more voluminous; they are occasionally found to have multiplied, and you may count two, or three nuclei, rarely more, in each node;[2] the cellular form becomes more distinct, owing to the thickening of the trabeculæ; the nerve-tubes appear to be further apart each from each—in reality, they have chiefly diminished in volume, and this kind of atrophy goes on at the expense of the medullary sheath, for the axis-cylinder has preserved its normal diameter, or it may even be hypertrophied. The amorphous matter which surrounds the fibres of the reticulum, on all sides, appears to be more abundant than in the healthy state.[3]

b. The nerve-tubes in the *second zone*, which may also be called the *transition zone*, have become still more slender. Many of them seem to have disappeared; in reality, they have been merely deprived of their medullary sheaths, and are now only represented by their axis cylinders, which, indeed, sometimes acquire comparatively colossal dimensions.[4] As to the trabeculæ of the reticulum, these offer not less remarkable alterations. They have become more transparent, their outlines are less distinct; finally, in certain parts, and this is a really fundamental fact, they are replaced by bundles of long and slender *fibrils*, closely analogous to those which characterize common connective tissue (laminous tissue). These fibrils are disposed in a direction parallel to the greater axis of the nerve-tubes; hence but little of them is seen in transverse sections, except their extremities, which present the appearance of a multitude of very fine dots. They tend, we have said, to usurp the place of the fibres or trabeculæ of the reticulum; but they, also, invade the meshes which contain the nerve-tubes, according as these diminish in size by loss of medullary matter, so that the reticulated or alveolar appearance which the connective gangue or matrix shows so distinctly in the healthy state, tends to become more and more effaced.[5]

c. The *central region* of the sclerosed patch, you are aware is that in which the most marked alterations are observed. Here all vestige of fibroid reticulum has disappeared; we no longer meet with distinct trabeculæ or cell-forms; the nuclei are less numerous and less voluminous than in the external zones; they are shrunken in every direction, appear shrivelled, and do not take so deep a tint as usual under the action of carmine.[1] They may be observed forming little groups here and there in the interspaces between the bundles of fibrillæ. The latter, however, have invaded every part. They now fill up the alveolar spaces, from which the medullary matter has completely disappeared. Nevertheless, a certain number of axis-cylinders, those last vestiges of the nerve-tubes, still persist in the midst of the fibrils; but they, in general, no longer retain that comparatively large volume they occasionally possessed in the early phases of the alteration; most of them, indeed, have even diminished to such a degree that they might be mistaken, so close is the resemblance, for the fibril filaments of new formation, from which, however, we shall soon learn how to discriminate them.

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[1] Charcot, 'Société de Biologie,' 1868.

[2] Occasionally some of these nuclei present towards their middle region an indentation which seems to indicate the beginning of scission.

[3] Frommann, 2 Theil, Pl. ii, fig. 1, and *passim*.

[4] Frommann, Charcot.

[5] Frommann 2 Theil, *loc. cit.*, P. iv, figs. 1, 2, 3.

Such, gentlemen, is the final term of the morbid process, in that form of sclerosis which engages our attention. And this indefinite persistence, as we may call it, of a certain number of axis-cylinders in the midst of parts which have undergone the extreme of fibrillary metamorphosis, is, you will be careful to remark, a character which seems proper to disseminated sclerosis. It is certainly not observed, at least to the same extent, in the other varieties of gray induration, whether we have to deal with descending spinal sclerosis, consecutive on lesions of the brain, or with that which, primarily occupying the posterior columns, is justly considered to be the anatomical *substratum* of progressive locomotor ataxia.

B. The results of the examination of longitudinal sections confirm, on the whole, the data which have been laid before you; I may, therefore, spare you any lengthy details, and confine myself to the following observations which will make you better acquainted with some aspects of the neoplastic fibril-formation. In sections of the kind mentioned, the characteristics of this tissue are well seen, and here you can best observe the longitudinal direction of the fibrils, their brilliant appearance which makes them resemble elastic fibres, and their arrangement in slightly undulating and ever parallel fascicles. On dilacerating these bundles, you will notice that the fibrils of which they are composed are extremely thin, that they are opaque and smooth, that they seldom divide and anastomose, whilst, on the contrary, they are frequently interwoven and entangled, so as to form a kind of felted tissue, and finally that they are scarcely tinted by the action of carmine (fig. 11). The latter characters sufficiently distinguish them from the axis-cylinders which, moreover, are generally larger, translucid, and never ramified. They may also be easily discriminated from the fibres of the reticulum, with which they are sometimes mingled, inasmuch as the latter are thicker, shorter, and their borders bristle with branching processes; finally, they differ from the elastic fibres which are so often found in common connective tissue by an important character; they swell up under the influence of acetic acid and form a hyaline transparent mass, which change does not occur in the case of elastic fibres.[1]

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[1] Frommann, Charcot.

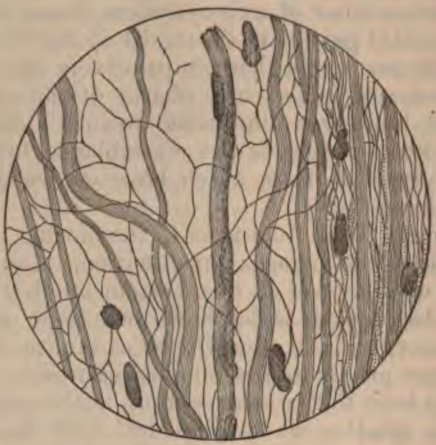

FIG. 11 represents a fresh preparation, taken from the centre of a patch of sclerosis, coloured with carmine, and dilacerated. In the centre is seen a capillary vessel, supporting several nuclei. To the right and left of this are axis-cylinders, some voluminous, others of very small diameter, and all deprived of their medullary sheaths. The capillary vessel and the axis-cylinders were vividly coloured by the carmine; the axis-cylinders present perfectly smooth borders, without ramification. Between them are seen slender fibrillæ of recent formation, which form on the left and in the centre a sort of network resulting from the entanglement or anastomosis of the fibrils. These are distinguished from the axis-cylinders, 1st, by their diameter, which is much smaller; 2d, by the ramifications which they present in their course; 3d, by taking no coloration from carmine. Nuclei are seen scattered about; some of them appear to be in connection with the connective fibrils; others have assumed an irregular form, owing to the action of the ammoniacal solution of carmine.

Can we proceed further in the study of the fibres, and endeavour to determine their mode of formation? Are they, for instance, as Frommann asserts, partially produced in the very substance of the fibres of the reticulum which they are destined soon to displace, and partially at the expense of the cells and the nuclei of the neuroglia? Do they arise, on the contrary, as others believe, either from the pre-existing amorphous matter, or from a newly formed blastema? In other words, is there metamorphosis or substitution? The question, we think, must remain undecided yet awhile; all that we can say respecting it is that the fibrils have sometimes seemed to take root in the substance of the nuclei or cells, and that this fact, if it were confirmed, might be cited in support of Frommann's thesis.

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[1] Valentiner, Zenker, *loc. cit.*; Vulpian 'Cours de la Faculté,' 1868.

I cannot pass over in silence the different alterations which those bloodvessels undergo that traverse the nodules of sclerosis. These changes may be well studied in the longitudinal sections of the cord, hardened by chromic acid. At the commencement, that is to say, in the peripheral zone, the parietes of these vessels, even of the finest capillaries, appear much thickened, and contain a larger number of nuclei than in the normal state. Nearer the centre of the nodule the nuclei are still more abundant, and, besides, the adventitious coat is replaced by several layers of fibrils quite similar to those which are simultaneously developed in the substance of the reticulum.[1] Lastly, at the final term of alteration, the walls of the vessels have become so thickened that their calibre suffers a notable diminution.[2]

I should notice, in passing, the habitual presence of a certain number of amyloid corpuscles in the midst of the fibrillary tissue. But I should at the same time mention the singular fact that these bodies are always less abundant in disseminated sclerosis than in the other varieties of gray induration.

C. It is not always without difficulty that we succeed in finding, in specimens which have not been prepared with chromic acid, all the details which I have just described. On the other hand, the fresh specimens offer this advantage, namely, they allow us to remark certain alterations which would have passed unnoticed if we confined ourselves to hardened preparations only. I allude here to the existence of globules and granulations of an apparently fatty or medullary nature, which we almost constantly[3] meet with in more or less considerable numbers in the substance of the sclerosed patches in the fresh state, and which soon disappear without leaving any trace when the preparation has been steeped some little time in chromic acid. Now, gentlemen, the presence of these fatty granulations is connected with an important phase of the morbid process, I mean with the destruction of the nerve-tube. However, before entering into a discussion of this subject, I think it useful to begin a little further back and to recall to your memory, by a succinct description, the modifications of structure which the peripheral nerves undergo when they are separated, by complete section, from the nervous centres.

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[1] Vulpian, 'Cours de la Faculté.'

[2] Frommann, *loc. cit.*

[3] This fact is, at least, mentioned by all the authors who have studied fresh specimens (Valentiner, Rindfleisch). It has not been absent from any one of the specimens which I have examined under similar conditions. See also Rokitansky in 'Bericht der Akademie der Wissensch. zu Wien,' t. xxiv, 1857.

At the outset I would remind you that, in the peripheral nerves the nerve-tubes are essentially constituted as in the spinal cord by a cylinder of medullary matter and by an axis cylinder, but that they also possess a sheath of connective tissue, the membrane of Schwann, which, according to the most recent researches,[1] does not appear to exist in the more slender tubes of the nerve centres, or at least only shows itself there in a rudimentary state.[2] You will perceive in a moment that this anatomical peculiarity, though apparently insignificant, is not devoid of interest from the point of view we occupy.

The following are the phenomena to which I wished particularly to call your attention. Eight or ten days after section of the nerve there supervenes a sort of coagulation of the medullary matter which breaks up into small masses, irregularly globular, with dark sinuous margins, showing a double outline, and having consequently preserved all the optical characters of myeline or medullary substance. Segmentation making new progress, in the following days, it will soon be perceived that Schwann's sheath no longer contains irregular masses of myeline, but globules presenting the appearance and the micro-chemical characters of fat. These globules, which are at first rather large, become gradually smaller and smaller as the process of division goes on, and finally they are replaced by very fine granulations, resembling dust, that fill the sheath of connective tissue. Mingled with these is found a certain proportion of paler granulations of a protein nature; and lastly, globules and granulations disappear, and Schwann's sheath, collapsing on itself, becomes so plaited and wrinkled that when you examine a certain number of such altered nerve-fibres, placed side by side in the field of the microscope, you would think you beheld a fascicle of filamentous connective tissue.

What becomes of the axis-cylinder during these changes? Composed as it is principally of proteine matter, it resists for a long time the action of the causes which have destroyed the medullary substance (or myeline), for it is occasionally still found in the sheath several weeks or even several months after section of the nerve.[3]

To sum up: under the new conditions of nutrition, in which the nerve-tubes are placed by section of the nerve, the medullary matter coagulates, then disintegrates and gives origin, on the one hand, to proteine molecules, and, on the other, to corpuscles which at first preserve the appearance of myeline, but which, in consequence of ulterior modifications, soon present all the characters of fatty granulations.[1]

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[1] Frey 'Handbuch der Histologie,' etc., 2e edit., p. 354; Leipzig, Schulte, 'De Retinæ Structurâ,' 1867, p. 22; Kölliker, 'Geweblehre,' 5e édit., 1867, t. iv, p. 257.

[2] Vulpian, 'Leçons sur la Physiologie,' etc., p. 316.

[3] See Vulpian, 'Leçons de Physiologie,' pp. 237 and 298; Rindfleisch, 'Lehrbuch der Pathologisch Gewebelehre,' pp. 10 et 20, 1866.

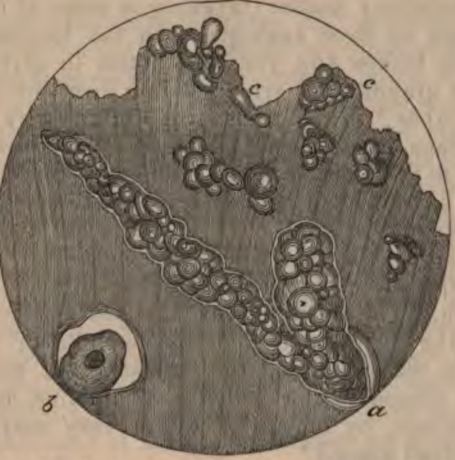

Fig. 12.—Patch of sclerosis in the fresh state: *a*, lymphatic sheath of a vessel distended by voluminous fatty globules; *b*, a vessel divided transversely. The adventitious coat is separated from the lymphatic sheath by a free space, the fatty globules which distended the sheath having disappeared; *cc*, fatty globules, gathered into small groups, dispersed here and there over the preparation.

Let us now return to the patches of sclerosis. Here we shall have to study phenomena closely analogous to those we have just been discussing.

In the substance of the sclerosed nodule, in fresh specimens, we almost constantly meet, as already mentioned, with globules or granules presenting the general appearance of fatty bodies. Their number is sometimes considerable. They show themselves under two principal aspects. Some constitute comparatively voluminous masses, whose dark and sinuous edges bound forms which sometimes represent irregular oval globules, sometimes are club-like, and occasionally kidney-shaped (Fig. 12). They present a double border like the myeline (or medullary matter), to which they also approximate in yet other respects. Other globules are true fatty droplets or granulations, sometimes free, sometimes aggregated so as to constitute confused heaps or coherent clusters, otherwise called *granular bodies*, devoid of nuclei and of enveloping membrane.[1] Proteine molecules are to be seen mingled here and there with these different granulations. All these products exactly resemble, you will observe, those which result from the disintegration of the medullary matter (myeline) in cases of nerve-section.

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[1] According to Robin, the myeline is a substance particularly rich in fatty principles, and it may, in this respect, be approximated to the contents of adipose cells ('Journal d'Anatomie,' 1868, No. 3, p. 309). Walter ('Virchow's Archiv,' 20, p. 426) has expressed the opinion that it is constituted by an amalgam or mixture of fatty and albuminoid bodies, which merely become dissociated in cases of degeneration of the nerve-tubes. See also Rindfleisch, *loc. cit.*, p. 20, § 52.

Let us follow up these analogies. In the longitudinal sections which I place before you, you will notice in certain parts long trails of fatty granulations arranged in a direction parallel to that of the nerve-tubes;[2] in the transverse sections they form here and there little heaps, like separate islets, which correspond with tolerable fidelity to the position of the alveolæ. Generally, indeed, the granulations have broken bounds, crossed the borders of the alveolæ, and become scattered over the adjacent tissues. But you cannot be surprised at this, when you remember that the nerve-tubes of the spinal cord are devoid of that cellular coat (Schwann's sheath) which, in divided nerves, incloses and contains all the products of the disintegration of the medulla or myeline. The meshes of the reticulum and the interstices of the fibrillæ offer easy ways of escape to the globules of myeline, as well as to the fatty granulations, by means of which they may permeate the tissue and spread abroad.[3]

Lastly, we would have you remark that the masses of (apparently) medullary matter and the fatty granulations are never met with in the centre of the sclerosed patch, that is, in the region where the work of fibrillary metamorphosis and of destruction of nerve-tubes has terminated; on the contrary they always occupy the external portions of the patch,[4] the peripheral and transition zones. Now, as you are aware, the morbid process is here in full activity; here it is that, compressed on all sides and strangulated by the thickening trabeculæ of the reticulum and afterwards by the fibrillary fascicles encroaching on the alveolæ, the medullary cylinder gradually diminishes in volume and then disappears altogether, leaving the nerve-tube at length only represented by the axis-cylinder. The accumulation of medullary or fatty globes and the destruction of the myeline-cylinder consequently take place simultaneously; we may even add that they proceed abreast, since the former ceases when the latter is concluded. The coexistence of the two phenomena evidently cannot be fortuitous, and, taking note of what precedes, it seems to us legitimate to conclude that the medullary and fatty corpuscles in question are nothing other than the wreck and detritus resulting from the disintegration of the nerve-tubes.[1]

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[1] Besides these granular bodies proper (*Fettkornchen Agglomerate*) may be found, in sclerosed patches, granular bodies having nuclei, that become coloured by the carmine test, and possess an enveloping membrane (*Fettkornchen Zellen*); these are simply cells of the neuroglia undergoing fatty degeneration. See, in reference to the distinction to be drawn between granular bodies, I. Poumeau, 'Thèse de Paris,' 1866; Rokitansky, 'Bericht der Akad. der Wiss. zu Wien,' t. xxiv, 1857; Wedl, 'Rudim. of Patholog. Histolog,' p. 292, London, 1855.

[2] It is not rare to meet, in the midst of the fibrillæ, with axis-cylinders partially denuded, to which globular masses adhere, at intervals, which have the appearance of medullary matter,

[3] Charcot, 'Société de Biologie,' 1868.

[4] *Ibidem.*

What becomes afterwards of these fatty granulations? They disappear in all probability by absorption; you know that no vestiges of them are discoverable in the central regions of the sclerosed patches. This is the place to submit to your notice a phenomenon which undoubtedly is connected with this act of absorption. In the preparations, which I am about to send round for inspection, you will observe that, in those parts where the products of nerve-disintegration are found, the lymphatic sheaths of the smaller vessels inclose within their cavities varying proportions of fatty granulations, or even, though more rarely, of corpuscles presenting the characters of myeline. In certain points, these different products are so abundant that the lymphatic sheaths are excessively distended; the vessels then appear to have swollen to twice and thrice their normal size, and they stand out, like little white tracks visible to the naked eye, on the gray ground of the sclerosed patch. Nevertheless, the coats of these vessels offer no other alterations than those indicated above, which certainly have no relationship with atheromatous degeneration. In short, we have here to deal with a fatty infiltration of the lymphatic sheaths, and not at all with a primary lesion of the vascular parietes. The same phenomenon is again met with in cerebral softening from arterial obliteration; in most of the forms of primary or secondary sclerosis; and, in short, in very different affections of the nervous centres, which, however, have this in common, that they all determine the fatty degeneration of the nerve-tubes. The true characters of this phenomenon appear to have been suspected by Gull,[2] and by Billroth,[3] but it has been principally cleared up by M. Bouchard, in his excellent studies on the secondary degenerations of the spinal cord.[4]

The description which has been given you of sclerosic alteration, disseminated in patches, relates chiefly to the white substance of the cord; but it may be equally applied, at least in a general way, to the gray matter. In both substances, in fact, the neuroglia is fashioned on the same model, and the alterations effected in it do not essentially differ; consequently, after the remarks already noted, I shall only make special mention of the modification which the nerve cells experience when, on the gray substance being invaded, they become comprised within the area of a sclerosed patch. These cells do not become the seat of nuclear proliferation, contrary to what under similar circumstances occurs in the cells of the connective tissue whose nuclei generally multiply; and this is, in fact, a characteristic which might, if needed, be a help to distinguish one from the other of these two orders of anatomical elements. The nerve-cells undergo a peculiar alteration which may be designated by the term *yellow degeneration*, on account of the ochreous tint which they assume, and which is occasionally somewhat intense; in this state they cease to be coloured by carmine as in the normal state; the nucleus and the nucleolus seem to be formed by a substance having a vitreous and brilliant appearance. It is the same as regards the body of the cell, which, besides, appears as if composed of concentric strata.

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[1] This opinion has been already very distinctly expressed by Rokitansky in 1858. 'Bericht,' etc., *loc. cit.*, 1857.

[2] "Cases of Paraplegia," 'Guy's Hospital Reports,' Third Series, 1858, t. iv.

[3] 'Archiv der Heilkunde,' 3 Jahr., p. 47.

[4] Bouchard, 'Archives Générales de Médecine,' Mars et Avril, 1866; Thèses de Paris, 1867, p. 44.

Finally, all parts of the cell are seized by atrophy, which may lead to a comparatively considerable diminution of its bulk, whilst, at the same time, the cell processes dwindle away and disappear.[1]

In the encephalon, and also in the optic and olfactory nerves, the sclerosed patches present essentially the same characters as in the cord; hence we do not think it would be of any advantage to enter into details in relation to them.

Now that we have reached the conclusion of our study, we may try to array, in their natural order of sequence, the phenomena which go to make up the alteration in question, and thus endeavour to determine the pathological method by which this morbid change is produced.

Undoubtedly, the multiplication of nuclei and the concomitant hyperplasia of the reticulated fibres of the neuroglia constitute the initial, fundamental fact, and necessary antecedent; the degenerative atrophy of the nerve elements is consecutive and secondary; it had already begun when the neuroglia gave way to the fibrillary tissue, though the wasting afterwards proceeded with greater rapidity. The hyperplasia of the vascular parietes plays merely an accessory part.

In what consists the affection of the neuroglia which marks the beginning of this series of derangements? It is easy to discover there all the characteristics of formative irritation. But, after recognizing the fact that disseminated sclerosis is a primary and multilocular chronic interstitial myelitis or encephalitis, it remains for us to determine the histological characters which distinguish it from other forms of sclerosis of the nerve-centres, and also from several kinds of myelitis or encephalitis which, having their starting-point likewise in the neuroglia, nevertheless do not issue in fibrillary metamorphosis. We will endeavour, at an opportune moment, to fulfil this duty. At present, gentlemen, we hasten to leave the department of pathological anatomy for that of clinical observation in order to show you by what array of symptoms disseminated sclerosis of the nervous centres makes its existence known.[1]

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[1] Frommann, *loc. cit.;* Vulpian, 'Cours de la Faculté,' 1868; Charcot, 'Société de Biologie,' 1868.

LECTURE VII.

DISSEMINATED SCLEROSIS: ITS SYMPTOMATOLOGY.

SUMMARY.—Different aspects of disseminated sclerosis, considered from a clinical point of view. Causes of error in diagnosis.

Clinical examination of a case of disseminated sclerosis. Tremor: modifications caused thereby, in the handwriting; characters which distinguish it from the tremor of paralysis agitans, chorea, general paralysis, and the motor incoördination of ataxia.

Cephalic symptoms. Disorders of vision: diplopia, amblyopia, nystagmus. Impeded utterance. Vertigo.

State of the inferior extremities. Paresis. Remissions. Absence of disorders of sensibility. Commixture of rare symptoms; tabetic phenomena; muscular atrophy. Permanent contracture. Spinal epilepsy.

IN the preceding lecture we minutely described the anatomical lesions of multilocular sclerosis of the nervous centres. Leaving aside, therefore, this portion of its history we shall proceed, to-day, to point out the series of symptoms by which it makes its existence clinically known.

I.

A. It is singular that a morbid state which possesses so distinct and so striking an anatomical substratum, and which, in short, is not a rare disease, should have escaped clinical analysis for such a length of time. Yet nothing is simpler, as I trust to show you, than to diagnose the affection in question, by the bedside of the patient, at least when it has reached its typical period of perfect development.

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[1] In a note published in the 'Archives de Physiologie' (1873, p. 753), one of Professor Charcot's students, Dr. Debove, has shown cause for the modification of the generally received opinion in reference to the histology of disseminated sclerosis. According to his researches, the sclerosed parts are formed of fibrillæ and of flat cells, quite similar to the cells of common connective tissue. He has succeeded in demonstrating this, through having employed the method of interstitial injections.

These facts are very different from what was believed with respect to the structure of the neuroglia (see note, p. 138), before M. Ranvier demonstrated that the connective tissue of the nerve-centres does not essentially differ from that of other organs; the only striking peculiarity being, according to M. Ranvier, the small diameter of the fibrillary fascicles.—(*Note to the Second French Edition.*)

If it be asked what cause so long deferred the recognition of disseminated sclerosis and its admission into nosological charts where it should occupy a place beside other better-known forms of primary sclerosis of the nervous centres, it is proper to point to the diversity of aspects under which it may be encountered in the hospitals. It is, in fact, an eminently polymorphic affection.

Our anatomo-pathological studies ought to have made you anticipate that it would be so. You remember that the patches or islets of sclerosis sometimes occupy the spinal cord exclusively, that in other cases they predominate in the cerebral hemispheres and the medulla oblongata, and that, finally, there are cases in which they are dispersed throughout all the departments of the nervous centres. These varieties of position induced us to recognize, from an anatomical point of view, the three following forms: 1st, the *cephalic form;* 2d, the *spinal form;* and 3d, the *mixed* or *cerebro-spinal form.* It was easy to foresee that each of these forms would be represented by a group of symptoms peculiar to itself.

B. Let us first, if you please, concentrate our attention on the cerebro-spinal form. It is, in truth, the most interesting in every respect and that which you will have occasion most frequently to observe in practice. Well, even when considered in this type, the disease may assume a variety of masks. Allow me, in support of this assertion, to mention an anecdote which one of my colleagues recently related to me.

A very distinguished physician, one, however, who was but little familiar with the symptomatology of disseminated sclerosis, had come to visit my friend in the clinical department over which he presides. In order to do him honour, my colleague presented him a case of the new disease,—a very fine specimen of the cerebro-spinal type The patient, leaving his bed, took a short walk down the ward. "This is an ataxic," said the visitor. "Perhaps so," replied my colleague, "but what do you think of the rhythmical movements by which the hand and upper extremities are shaken?" "True," said the visitor, "he is also affected with chorea, or perhaps with paralysis agitans." The patient was then questioned. He replied, but, in replying, showed a marked difficulty of enunciation; he scanned the syllables in a very peculiar manner; and the utterance of a word was often preceded by a slight trembling of the lips. "I understand," said the physician, "you wished to puzzle me by presenting a most complicated case. Here are symptoms which belong to general paralysis. Pray don't proceed any further;

your patient probably is a living conpendium of all nervous pathology."

Now, gentlemen, I repeat it, this was simply a case, though a very complete one, of the cerebro-spinal form of disseminated sclerosis.

C. Paralysis agitans is especially the disease with which this form of sclerosis has been the most persistently confounded, and for which it is, undoubtedly, still the most frequently mistaken. It was because of this confusion that, at the time we laboured to draw forth disseminated sclerosis from the chaos of chronic myelitic affections, we urged M. Ordenstein, then one of our students, to tabulate in parallel columns the opposite characters that divide this disease from paralysis agitans, for the better understanding of the contrast.[1] How M. Ordenstein acquitted himself of this duty is known to you, and I do not hesitate to declare that his dissertation marks a serious progress in the clinical history of chronic diseases of the nervous system.

In recent days, Herr Baerwinkel, a distinguished physician of Leipzig, after having related a very interesting example of cerebro-spinal sclerosis, which, however, presented no tremor (as sometimes happens), seems to insinuate that M. Ordenstein was pleased to create difficulties which had no real existence, in order to give himself the facile satisfaction of surmounting them. According to him, there is no analogy whatever between the two diseases. Dr. Baerwinkel must, doubtless, have forgotten that in 'Canstatt's Jahresbericht' he gave, some ten years ago, the analysis of a case observed under Skoda's care,—in that case, paralysis agitans had been diagnosed during life, whilst, on post-mortem examination, patches of disseminated sclerosis were found in all parts of the cerebro-spinal axis. The case appears to have been noted with the greatest fidelity. It is stated, and this point deserves remark, that the tremor, contrary to what occurs in ordinary cases of paralysis agitans, only showed itself when voluntary movements were made, and subsided when the patient was at rest.[2]

Nor can Herr Baerwinkel have overlooked the case recorded by Herr Zenker in Henle's Journal. Here again the existence of multilocular sclerosis was only revealed by the post-mertem examination.[3] During life, Professor Hasse had made a diagnosis of paralysis agitans, and yet, in the symptomatological description, there is stress laid on the nature of the tremor, which only showed itself under the influence of emotion or on the occasion of voluntary movements.

These examples suffice, I presume, to show you that, in spite of the opinion of Herr Baerwinkel, it is possible to confound the two diseases, since such confusion has been committed by clinical observers whose skill is above all question.

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[1] "Sur la Paralysie Agitante et la Sclérose en Plaques Généralisée," Paris, 1867.

[2] 'Vein. Med. Halle,' 13, 1862.

[3] Zenker, 'Zeitschrift für Medizin,' Band iii, Reihe, 1865, p. 228.

That being established, I am ready to concede that the different disguises assumed by disseminated sclerosis are coarse masks, and that to-day, when recent works[1] have illuminated the field of diagnosis, it is scarcely permissible to be caught in the snare. But it is time, gentlemen, to place you in a position to distinguish the characters by means of which cerebro-spinal disseminated sclerosis may be separated from those diseases which more or less closely resemble it.

II.

You are not unaware, gentlemen, of what value you must set on clinical descriptions, eloquently detailed at a distance from the bedside of the patient. They seldom succeed, whatever the effort, in doing more than giving origin to indistinct images which generally leave but a vague and transient impression on the mind of the auditor.

In order to avoid, as much as possible, falling into the fault I have just mentioned, I will proceed in your presence to the methodical examination of a patient who presents all the symptoms of the cerebro-spinal form of disseminated sclerosis, in the period of perfect development.

Mademoiselle V—, aged 31, has been suffering for about eight years under the affection which forms the object of the present study. Admitted to La Salpêtrière three years ago, she was bequeathed to me by M. Vulpian when he left this hospital, and he, at the same time, gave me, in reference to her case, a detailed and most valuable note. The invasion of the disease dates, we have said, from eight years ago; it is, therefore, a case of old standing. I will tell you afterwards of the different changes which characterized the early phases of the evolution of symptoms. For the moment I wish to confine myself to an analysis of her actual condition.

One symptom which, doubtless, struck you all from the first on seeing the patient enter, assisted by a nurse, was certainly the very special rhythmical tremor by which her head and limbs were violently agitated whilst she was walking.

You have likewise noticed that when the patient sat upon a chair, the tremor disappeared at once and completely from her upper and lower limbs, but only partially from the head and trunk. I lay stress on this latter point, whilst calling your attention to the fact that the new attitude, assumed by the patient, is far from being one of absolute rest as regards the muscles of the body and neck. Besides, we must make allowance for the existence of emotion which undeniably plays a certain part here. I shall have occasion to show you Mademoiselle V— when reclining in bed, and in complete repose; you will then be able to assure yourself of the utter absence of all trace of tremor in the different parts of her body. To cause the rhythmical agitation again to appear throughout the body, it will suffice to make the patient rise from her seat. To bring it back merely in a partial manner, in one of the upper extremities for instance, I will request her to lift a glass full of water, or a spoon, to her mouth. You can see that, in the several acts prescribed by the will, the tremor increases in direct ratio with the extent of the movement executed. Thus, when the patient wishes to lift a glass full of water to her lips, the rhythmical agitation of the hand and forearm is scarcely noticeable when taking hold of the object; but it becomes more and more exaggerated as the glass is brought nearer to the eyes; and at length proceeds to such an extent that, at the moment when the goal is being attained, the glass is, as you observe, dashed with violence against the teeth, and the water is flung out to a distance. This great disorder is not shown, I repeat, save in the performance of movements of a certain amplitude. As regards petty operations, such as sewing or ravelling linen, the oscillations, on the contrary, are almost null. Some time ago, the patient could still write distinctly enough; the letters, indeed, were tremulous, but perfectly readable.[1]

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[1] Bourneville et L. Guerard, "De la Sclérose en Plaques Disséminées," Paris, 1869; Bourneville, "Nouvelle Étude sur quelques Points de la Sclérose en Plaques Disséminées," Paris, 1869.

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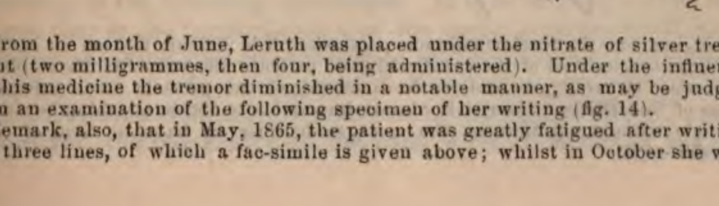
[1] We give below two specimens of the handwriting of a patient named Leruth, who succumbed to disseminated sclerosis in M. Charcot's wards. This woman was admitted to La Salpêtrière September 24, 1864. In May, 1865, M. Charcot obtained this fragment of the writing (fig. 13).

Fig. 13.



From the month of June, Leruth was placed under the nitrate of silver treatment (two milligrammes, then four, being administered). Under the influence of this medicine the tremor diminished in a notable manner, as may be judged from an examination of the following specimen of her writing (fig. 14).

Remark, also, that in May, 1865, the patient was greatly fatigued after writing the three lines, of which a fac-simile is given above; whilst in October she was

To sum up, *the tremor* in question *only manifests itself on the occasion of intentional movements of some extent; it ceases to exist when the muscles are abandoned to complete repose.* Such, gentlemen, is the phenomenon which I have been led to regard as one of the most important clinical characters of cerebro-spinal disseminated sclerosis. I do not, indeed, pretend to put this forward as a pathognomonic symptom; I am not unaware that a tremor showing itself, with somewhat similar characters, is occasionally observed in affections other than disseminated sclerosis, as, for instance, in mercurial poisoning, in chronic cervical meningitis with sclerosis of the cortical layer of the cord, in primary or consecutive sclerosis of the lateral columns, etc. It is not, as we shall see, a constant symptom. But what I wish at present to place prominently before you is the fact that, in disseminated sclerosis, when no other complication supervenes, the tremor, if it exist at all, presents itself always with the characters which I have assigned to it. In some this is a symptom which, by itself alone, would suffice to distinguish multilocular sclerosis of the nervous centres from some affections which so nearly resemble it as to render confusion possible. In reference to this subject I shall enter into some details.

The tremor of *paralysis agitans* exists as well when the members are in a state of repose as when they are set in motion by the will. I present a patient in whom the tremor has persisted for long years, without cessation or truce, during the patient's waking

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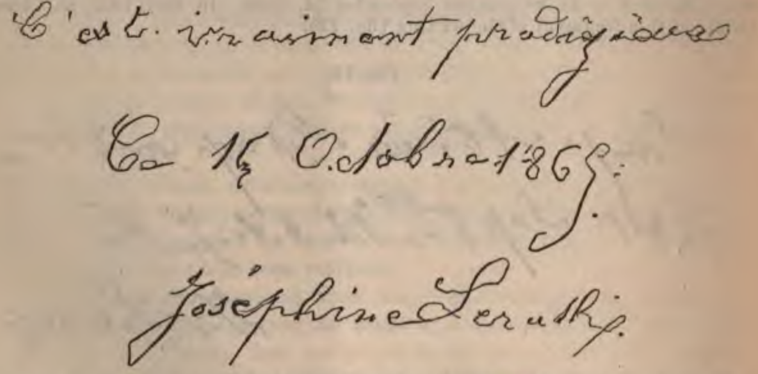
able to write a dozen lines with ease. We have selected, for the second specimen, the first and last lines of what she wrote.

FIG. 14.



C'est un aimant prodigieux

Le 15 Octobre 1869.

Joséphine Lerudy.

Judging from the specimens in our possession, it is difficult to form an opinion on the characteristics of the handwriting of patients affected with disseminated sclerosis. Generally, indeed, we have examined the patients at an advanced stage of this disease; then it is almost impossible to obtain anything beyond a scribble without significance, the more so because we have no term of comparison.—(B.)

hours. It never stops save when this unfortunate woman is plunged in profound sleep. There are cases of shaking palsy where the tremor only shows itself intermittently, but, singularly enough, it is just in such cases that the tremor shows itself rather when the limbs are at rest, and ceases when they are set in motion by the will. You can perceive in another patient, whom I submit to your observation, this peculiar characteristic of paralysis agitans. Remark, also, in both these women, that the head takes no share in the trembling; or, if it seem shaken by the oscillations, these are plainly communicated to it by the agitation of other parts of the body,—there is transmission of shocks from the affected members and trunk. The absence of tremor of the head seems to me an almost constant fact in shaking palsy. I will add that in this affection the jerks are of much less extent, more regular, rapid, and serried, if I may so speak, than in multilocular sclerosis. In the latter, the oscillations are larger, and resemble, in many respects, the gesticulations of chorea; this analogy is so close that before the publication of the works which have caused it to be admitted into clinical lists, disseminated sclerosis has been sometimes designated under the names of rhythmic chorea and choreiform paralysis.

It is, however, always easy to distinguish the odd and disorderly movements of *chorea*, properly so called, from the rhythmical oscillations of multilocular sclerosis. Note, firstly, that in the latter case, if the action of the upper extremity when lifting an object to the lips be considered, the *main direction of the motion persists in spite of the obstacles caused by the jerks of the tremor*, which, as we have just said, augment as the hand approaches its goal. In chorea, on the contrary, the *main direction of motion would be disturbed from the outside by contradictory movements*, quite disproportionate in magnitude, *which cause the goal to be missed.* Add to this that the movements of chorea show themselves suddenly and unexpectedly, when the limbs are in a state of perfect rest; thus, apart from any act of the will, the choreic patient is seen to thrust out his tongue, make a grimace, or abruptly raise a limb, etc. Now, such things are altogether unknown in multilocular sclerosis.

When, in *progressive locomotor ataxia* (sclerosis of the posterior columns), the upper extremities are affected, we find, as regards purposed acts, incoördinated movements which, to some extent, recall the gesticulation of chorea, and the jerks of multilocular sclerosis. The danger of confounding them may be avoided by attending to the following characters. It is to be observed, at the outset, that in the incoördination of the ataxic patients we do not, properly speaking, find any tremor or rhythmical jerks, but rather gesticulations of different degrees of disorder, abruptness, and extent. Examine studiously, in the case of the patient whom I place before you, the movements of the hand when in the act of taking hold of a small object, and you will find truly characteristic peculiarities. You see how, at the moment of grasping, the fingers separate excessively, and are extravagantly extended, bending towards the back of the hand. Then the object is seized suddenly, with a dash, in an almost convulsive manner by the abrupt and disproportionate flexion of all the fingers. This is a symptom of ataxia; you will never observe anything of the kind in disseminated sclerosis. Lastly, I would add—and this final trait is truly decisive—that, in ataxia, the closing of the eyes has always the effect of exaggerating in a very marked manner the incoördination of the movements, whilst it does not at all modify the rhythmical jerks of multilocular sclerosis.

We should not, however, forget that some of the symptoms of ataxia are found occasionally mixed up with those of disseminated sclerosis, when the sclerosed islets in certain regions of the cord spread over a certain height of the posterior columns. A case, the history of which may be found recorded at length in Cruveilhier's 'Atlas d'Anatomie Pathologique,' may be cited as an example of this class.[1] It is the case of the patient Paget. In order to grasp and use a pin she required to have her eyes open, otherwise the pin dropped from her fingers. On a post-mortem examination, it was found that one of the sclerosed patches occupied a considerable extent of the posterior columns in the cervical enlargement of the cord. But I shall not now dwell at any greater length upon this point, to which we shall have several opportunities of again referring

We have hitherto, almost exclusively, studied the question of tremor in its connection with the upper limbs; but we also know that it may affect the head, the body, and the lower extremities. It presents itself, in these different parts, with all the characters that we have described in reference to the upper limbs, that is to say, it is absent in repose, and shows itself on the occasion of purposed movements, or in case of attitudes which cannot be maintained except by the active and more or less energetic tension of certain muscles or sets of muscles.

In order to complete the characteristics of this symptom, we must enter into a few details. Tremor, gentlemen, as I long ago declared, is an almost constant symptom in the cerebro-spinal form of disseminated sclerosis. It must not be forgotten, however, that exceptional cases exist, in which—though the fact is as yet inexplicable—no tremor presents itself amongst the symptomatological group. I have myself observed several cases of this kind. But you should note, gentlemen, that tremor may have existed, to a greater or less extent, at a certain anterior epoch of the disease, and may have disappeared at the time when the patient offers himself for examination. It is, therefore, necessary, on this account, to question with the greatest care those patients in whom this symptom is apparently absent.

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[1] Cruveilhier, 'Atlas d'Anatomie Pathologique,' livraison 38, pp. i et ii.

It is the rule that the tremor disappears when the members are immobilized by permanent contracture, at a more or less advanced period of the disease. Though the tremor sometimes shows itself almost from the very beginning, yet it must be acknowledged that it is usually a late symptom. In conclusion, gentlemen, it is very frequent and almost customary that the tremor shall not last as long as the disease; it grows less marked as the patients decline in strength, and it sometimes completely vanishes before the fatal end arrives.

III.

You are now acquainted, gentlemen, with one of the most original and most important symptoms of sclerosis in generalized patches. A deeper and more circumstantial study of the case which we have before us will enable us to collect many other indicia which are not less valuable. We shall discover in our patient a whole group of symptoms, which I propose to call *cephalic*, as opposed to *spinal* symptoms. This group comprises certain disorders of vision, of speech, and of intellect.

A. Let us first apply ourselves to the question of visual disorders. These are diplopia, amblyopia, and especially nystagmus.

a. Diplopia, as happens also in locomotor ataxia, is an initial symptom, usually quite transient, but yet deserving of passing notice.

b. Amblyopia, on the other hand, is a persistent, and indeed a more frequent symptom of cerebro-spinal disseminated sclerosis. I believe I may affirm that, contrary to what takes place in posterior sclerosis, it very rarely issues in complete blindness.[1] This is a peculiarity worthy of notice, especially if you remember that patches of sclerosis have been found, after death, occupying the whole thickness of the nerve-trunk, in the optic nerves, in cases where, during life, an enfeeblement of sight simply had been noted.[2] This apparent disproportion between the symptom and the lesion constitutes one of the most powerful arguments which can be invoked to show that the functional continuity of the nerve-tubes is not absolutely interrupted, although these, in their course through the sclerosed patches, have been despoiled of their medullary sheaths and reduced to axis-cylinders.

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[1] In a case reported by M. Magnan ('Archives de Physiologie,' t. ii, p. 765) there was papillary atrophy of both eyes, with complete blindness.

[2] Case of the patient, Aspasie Byr, communicated by M. Vulpian. This observation is recorded, *in extenso*, in a work by M. H. Liouville, entitled "Observations détaillées de deux cas de sclèrose en ilots multiples et disséminés du cerveau et de la moelle épinière" ('Mémoires de la Société de Biologie,' 1868, p. 231).

On examining the eyes with the ophthalmoscope, an operation which is generally rendered difficult by the existence of nystagmus, we usually find, under such circumstances, either almost complete integrity of the papilla of the optic nerve, even when amblyopia is far advanced, or a partial lesion; or, finally, in the rare cases where blindness is complete,[1] total atrophy (marked by a pearly white coloration, and extreme tenuity of the vessels) with or without excavation of the papilla.

In the case of Mademoiselle V— we have simply a rather marked amblyopia of both eyes. No well-determined lesion has been discovered on ophthalmoscopic examination. It is worthy of notice that, in this case, flashes of light and sparks preceded the enfeeblement of sight. I have observed the same phenomena in several other cases of amblyopia connected with multilocular sclerosis.

c. *Nystagmus* is a symptom of sufficiently great importance in diagnosis, since it is to be met with in about half the number of cases. It is not found, so far as I know, in locomotor ataxia, save in very exceptional cases. You may observe that it exists, to a very advanced extent, in Mademoiselle V—. We have there, as you see, a quantity of little jerks or twitches, which cause the eyeballs to oscillate simultaneously from right to left, then from left to right, or inversely. There are cases in which the nystagmus is not present so long as the gaze is fixed on nothing, but shows itself suddenly, in a more or less manifest manner, as soon as the patients are asked to look attentively at any object.

B. There is a symptom more frequently found than nystagmus, one which is almost constant in multilocular cerebro-spinal sclerosis, since it is noted in twenty two out of the twenty-three cases that we have analyzed, and this is a *peculiar difficulty of enunciation* which you can study in our patient, where it exists in a typical state of perfect development.

The affected person speaks in a slow, drawling manner, and sometimes almost unintelligibly. It seems as if the tongue had become "too thick," and the delivery recalls that of an individual suffering from incipient intoxication. A closer examination shows that the words are as if measured or scanned: there is a pause after every syllable, and the syllables themselves are pronounced slowly. The patient hesitates in the articulation of his words, but there is, properly speaking, nothing like stammering. Certain consonants, *l*, *p*, and *g*, are peculiarly ill-pronounced.

There exists in the case of Mademoiselle V—, as you may observe, a certain slowness in the movements of the tongue, you see that it is even affected by very manifest tremulation when protruded. It must not be supposed, however, that this is a constant phenomenon, for I have several times found that speech might be impeded to a very great extent without the tongue presenting the least trace of tremor. The tongue always, at least according to my experience, preserves its normal volume, and I have never seen it wrinkled on the surface, as may be noticed in certain cases of labio glosso-laryngeal paralysis with atrophy of the lingual muscles.

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[1] Case quoted by M. Magnan.

The difficulty of enunciation, at first scarcely perceptible, becomes gradually aggravated during the course of the disease, until the patient's discourse is rendered nearly incomprehensible. In some instances it becomes suddenly aggravated, as if in paroxysms, and then grows temporarily better.

On the whole, the difficulty of speech which is observed in cerebro-spinal sclerosis approximates, in many respects, to the corresponding symptom of progressive general paralysis. I even think that, in many cases, it would be almost impossible to distinguish between them, if abstraction were made of the assistance given by concomitant phenomena. Add that the approximation may be rendered still closer by the circumstance that, in multilocular sclerosis, as in general paralysis, the utterance of words is sometimes preceded, as you can verify in our patient, by a slight and, as it were, a convulsive contraction of the lips.

However it be, this trouble in the articulation, to which I call your attention, is a very important symptom of multilocular sclerosis. It may potently contribute to settle the diagnosis, principally in those cases, which are indeed exceptional, where tremor of the hand and upper extremities is absent.

To this symptom may successively be added, especially in advanced stages of the disease, certain disorders of deglutition, of circulation, and even of respiration. These are symptoms of *progressive bulbar paralysis*, which ought to give the alarm, because, becoming rapidly aggravated, they have sometimes suddenly and almost unexpectedly determined the fatal termination. On account of the interest attaching to them in prognosis, they shall form the object of a special study.

C. *Vertigo*, in about three-fourths of the cases, is one of the phenomena which mark the invasion of multilocular sclerosis of the nervous centres. As far as I can judge from the descriptions given me by the patient whom I have questioned, the vertigo is generally of the gyratory kind. All objects seem to be whirling round with great rapidity, and the individual himself feels as if revolving on his axis. Threatened with loss of equilibrium, the patient lays hold of whatever is nigh him. In most cases, this giddiness returns in fits of short duration; sometimes, however, it persists almost without interruption for a certain period, superadded to the tremor and paralytic state of the members; it often contributes considerably to render it almost impossible for the patient to stand erect or continue his titubating walk. You must take care not to confound this titubation with the uncertainty of gait which is connected with diplopia; the latter ceases when the patient keeps one of his eyes closed.

The vertigo in question is all the more interesting because it belongs neither to locomotor ataxia, nor to paralysis agitans, and may consequently help in forming a diagnosis.

D. Most of the patients affected by multilocular sclerosis, whom I have had occasion to observe, have presented at a certain stage of the disease a truly peculiar *facies*. The look is vague and uncertain; the lips are hanging and half-open; the features have a stolid expression, sometimes even an appearance of stupor. This dominant expression of the physiognomy is almost always accompanied by a corresponding mental state, which deserves notice. There is marked enfeeblement of the memory; conceptions are formed slowly; the intellectual and emotional faculties are blunted in their totality. The dominant feeling in the patients appears to be a sort of almost stupid indifference in reference to all things. It is not rare to see them give way to foolish laughter for no cause,[1] and sometimes, on the contrary, melt into tears without reason. Nor is it rare, amid the state of mental depression, to find psychic disorders arise which assume one or other of the classic forms of mental alienation.

One of the patients of Valentiner, usually subject to melancholia, was, from time to time, seized with ambitious mania. A man, whose case has been recently recorded by Dr. Leube[2] looked upon himself as destined to become a king, nay, an emperor; he boasted that he possessed a large number of oxen, horses, and beautiful mansions. He was soon, he said, about to form a matrimonial alliance with "a countess," etc.[3]

Mademoiselle V— was seized, a few weeks ago, with a genuine fit of lypemania. She had hallucinations of sight and hearing. She beheld frightful apparitions, and heard voices threatening her with the guillotine. She was convinced that we wanted to poison her. During twenty days she refused all kinds of nourishment, and we were forced, during the whole of that time, to administer food by means of the stomach pump. To-day, these accidents have almost entirely vanished. Nevertheless, the voices are still heard from time to time. You see the patient has been taken, during our examination, with convulsive laughter which she cannot moderate, and which will soon be followed by a shower of tears.

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[1] A patient, under M. Charcot's care, of whom we shall have occasion to speak again, Hortense Dr—, is frequently seized with causeless fits of laughter, which she cannot control. Having been subject, before the invasion of the disease, to fits of anger, she has noticed, with regret, that they have increased since that period. (B.) On the other hand, one of two patients, whose cases I had an opportunity of studying, in Professor Behier's wards in the Hôtel Dieu, did not exhibit any marked intellectual disorder, although she had been many years in hospital. The second patient, whose symptoms were more advanced, seemed to wake from a dream, when spoken to, then, trying vainly to fix his gaze on the speaker, he answered intelligently, but the (gradually increasing) difficulty of enunciation made conversation painful. (Sigerson.)

[2] "Ueber multiple inselförmige Sklerose des Gehirns und Rückenmarks' ('Deutsch. Archiv,' 8 Bd., 1 heft, Leipzig, 1870, p. 14).

[3] One of the patients, Aspasie B—, observed by M. Liouville, in M. Vulpian's wards, had hallucinations; Rosine Spitale, whose history we have abridged (Bourneville et Guérard, *loc. cit.*, p. 92 from M. Valentiner, fell into stupor some months before the fatal termination of the disease. (B.)

IV.

In order to conclude the descriptive study of the case which I have presented you, gentlemen, as a type of *multilocular sclerosis* of the nervous centres, it only remains for me to direct your attention to the state of the lower extremities. You have seen that Mademoiselle V— cannot rise from her seat, stand erect, or attempt to walk, if she be not strongly supported by two assistants. It is easy to note that the cause of this motor impotence is, principally, a pseudo-tetanic rigidity which has seized on the lower extremities, and which, though very marked when the patient is seated or reclining, becomes exaggerated to the highest degree when she attempts to rise or walk.

This contracture of the lower limbs, at present permanent, only manifested itself quite recently in the case of Mademoiselle V—; it is, in fact, a symptom of the advanced stages of the disease. In the evolution of the morbid process it is always preceded at a considerable distance by a *paretic state*, presenting some peculiar features, with which I will first endeavour to make you acquainted.

In reference to this particular point the clinical history of Mademoiselle V— has been traversed by certain incidents which, without being exactly exceptional, still do not constitute the rule. Consequently I am forced to put it aside for the moment, reserving the right of soon returning to it. In the following description I will draw upon details recorded in a certain number of cases which I have collected, and in which the paretic period was developed in accordance with the normal conditions.

Paresis of the limbs.—We have here a more or less marked decline of the motor power of the limbs, which is frequently manifested at the very outset of the disease, and which is not usually connected with any notable disturbance of sensibility.

Generally one of the lower limbs is first and solely affected. It feels heavy and difficult to move; the foot turns at the least obstacle in walking, or the whole limb suddenly gives way under the weight of the body. The other limb is seized, sooner or later, in its turn; however, as the paresis advances with extreme slowness, the patients are still able, for yet a long while, to walk about with more or less ease and to attend to their occupations, but at last the day comes when, owing to an aggravation of the motor paralysis, they may be confined to bed. The upper extremities are themselves invaded, either simultaneously or one after the other, usually at a period far removed from the invasion of the disease. Frequently in the commencement there are remissions; thus, it is not rare to see the enfeebled lower limbs resume, for a time, their original energy. Such remissions may even occasionally take place two or three times. I point out this peculiarity to your notice because it certainly is not found, to the same extent, in other chronic diseases of the spinal cord.

I should revert for a moment, in order to lay stress on the fact, already noticed, of the absence of disorders of sensibility. The patients do, indeed, sometimes complain of formications, and of a feeling of numbness occupying the enfeebled limb; but these symptoms are usually transient and but little marked. Besides, it is easy to ascertain that cutaneous sensibility, in the affected members, is almost always preserved, in all its modes. The girdling pains, the fulgurant crises, which play so prominent a part in the early stages of progressive locomotor ataxy, are absent here. It is the same thing with respect to that loss of the sense of position of parts, which also belongs to ataxia. This does not occur in regular multilocular sclerosis, and patients affected by the latter disease can, with closed eyes, determine with exactness the position which has been given to their limbs. Nor has the closure of the eyes any marked influence on the power of the patient to hold himself erect, or on his manner of walking. His gait is uncertain, embarrassed, titubating, on account both of muscular weakness and of the tremor which, sooner or later, is superadded; the feet, held apart in order to enlarge the basis of support,drag awkwardly over the ground, from which it is hard to raise them. When titubation is very much marked the patients threaten to fall at every step, and they do, in fact, frequently come to the ground. The lower extremities are not flung forward, in an abrupt manner and convulsively, as we so commonly see them in sclerosis of the posterior columns. The sphincters are very rarely affected by the weakness which invades the muscles of the limbs, and this contrasts with what occurs in many spinal affections, where you see, at a very early stage, vesicular and rectal troubles superadded to the other symptoms. Finally, to complete the picture, we should lay stress on the habitual absence of trophic disorders of the muscles in the paraplegia connected with multilocular sclerosis. The enfeebled muscles preserve almost to the last their prominence and firmness; tested by faradaic exploration they do not present, at any stage, traces of notable enfeeblement of electric contractility.

Intermixture of unusual symptoms.—I made mention, as we proceeded, of a certain number of symptoms which I took care to eliminate, because they do not belong to the regular type of the disease. It is necessary to inform you now, by way of corrective, that these symptoms do intermingle, in certain cases, with the ordinary phenomena of multilocular sclerosis, and even become so very prominent that an observer, if not forewarned on the subject, would perhaps be almost necessarily mistaken. Under this aspect, the record of Mademoiselle V— may furnish us with valuable information. I extract, therefore, some details from it, dated March 24, 1867, that is to say, over three years ago. At that period, when, indeed, the paresis and tremor were so far advanced in the lower limbs as to make it impossible for the patient to walk, except by the help of two assistants, the following symptoms were noted: Whilst walking, the feet are slightly thrown forward, "as with ataxic patients." When the eyes are closed there is "exaggeration of the titubation, loss of equilibrium, and the patient would certainly fall if not strongly upheld by two assistants." In the lower limbs "tactual sensibility has diminished in a marked manner. The patient, with closed eyes, cannot tell what position has been given to her limbs. She experiences in them, from time to time, violent paroxysms of fulgurant pains." Finally, the existence of a girdling pain has been noted.

You have recognized, in this enumeration, nearly the whole series of phenomena which serve clinically to characterize progressive locomotor ataxy. Some of them are to be found present today in our patient, but they appear, generally speaking, in a very attenuated form, or relegated to the background. Do we mean to say that, even at the time when they seemed to predominate, they were of a nature seriously to embarrass the diagnosis? No, decidedly not, and I am convinced that, in all cases of the kind, you could avoid deception by bearing in mind the following observations:—

The very fact of paresis of the lower limbs (which does not exist in posterior sclerosis, or which, at all events, only shows itself at an advanced stage) being found mixed up with the *ataxic symptoms*, should put you on the true path. If it have preceded them the case is still clearer. You will also certainly have to chronicle the coexistence of some of the symptoms which belong only to multilocular induration, namely—tremor of the extremities, impeded enunciation, vertigo, nystagmus, etc. It is necessary, besides, to clearly understand the reason why ataxic symptoms are sometimes manifested in the course of multilocular induration, as I announced above. There is here in my opinion, no question of a combination of the elementary forms of two diseases—progressive locomotor ataxia and cerebro-spinal disseminated sclerosis. As for myself, I have never, in a post-mortem examination, met with the coexistence of multilocular gray induration and posterior *fasciculated* sclerosis; and, without denying that such an association could exist, I believe it to be at least infinitely rare. It is, on the contrary, common enough for the sclerosed patches (which, as a rule, principally occupy the antero-lateral columns) to cross the postero-lateral fissures and encroach on the posterior columns. Occasionally even, I have seen them, when they were confluent, involve a large portion of the substance of these columns throughout the whole extent of one of the regions of the cord, the lumbar region for instance. Now, in all cases of this kind, ataxic symptoms were manifested to different degrees of intensity during life. I have no doubt but that a similar arrangement will one day be found to account for the fulgurant pains, the motor incoördination, and, in a word, for all the phenomena of the same order which are stated in the record of Mademoiselle V—.[1]

Unusual symptoms of another kind may also be superadded to the regular symptoms of multilocular sclerosis. In several cases, which were otherwise well characterized, I have seen an atrophy of certain muscles, or groups of muscles, supervene, which recalled, both by its position and its mode of invasion, progressive muscular atrophy. I have twice had the opportunity of ascertaining the anatomical cause of this new complication; in both cases the irritative process, of which the sclerosed foci are the seat, had, in certain regions of the cord, extended to the nerve-cells of the anterior cornua of the gray matter, and these cells had, in consequence, undergone great alterations. Now, according to the researches which I have detailed to you, it is but little doubtful that progressive amyotrophy, whether protopathic or consecutive, most frequently arises from an irritative lesion of the great nerve-cells, termed motor cells.[1]

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[1] Cases of disseminated sclerosis, in which the posterior columns were involved so as to occasion some of the symptoms of locomotor ataxy, are numerous enough. We may mention, first, the case of Paget, recorded by Cruveilhier in his 'Atlas;' then the three cases which were related at length in our memoir. The first is that of the woman Broisat (disseminated sclerosis, principally occupying the posterior columns), who succumbed in M. Charcot's wards; the two others, which were perhaps more characteristic, inasmuch as the symptoms and lesions of locomotor ataxia were more prominent, were quoted from Friedreich. Finally, we will briefly summarize another case, which we noted during the siege, in M. Marrotte's wards:

Josephine Leg—, aged forty-six years, a silk-winder, has been suffering for two years. She presented the following ataxic symptoms—difficulty of walking with closed eyes; notion of position, with respect to lower limbs, greatly lost: frequent fulgurant pains in the knees and legs: girdle pains. But, along with those symptoms, these were noted, *i. e.*, considerable paralytic enfeeblement of the lower limbs; preservation of the different modes of sensibility in the upper and lower extremities; visual integrity. This woman succumbed to pyelo-cystitis, complicated with sacral eschars. *Autopsy:*—Sclerosed patches on the left external motor oculi and on the optic nerves; sclerosed patches on the pons Varolii, the right superior crus cerebelli, etc.; sclerosed patches on the surface of the lateral ventricles, in the interior of the centrum ovale, on the anterior face of the bulbus rachidicus, and in the fourth ventricle. In the spinal cord we found, 1st, a sclerosed patch, four inches long, occupying the left posterior column; 2d, another of less length and breadth on the right posterior column; 3d, beneath it, another rather circumscribed patch occupying both posterior columns; and 4th, on the antero-lateral surfaces of the cord, many small patches of sclerosis. (B.)

Permanent contracture of the limbs. Spinal epilepsy.—It is time now to revert to the contracture noticeable in the lower extremities of the patient V—, which, at present, constitutes a permanent phenomenon that you may study as a most perfect type. This, gentlemen, is an habitual symptom of the advanced phases of multilocular sclerosis. It does not follow on paresis, suddenly and without transition. At a certain stage of the paretic period there supervene, either spontaneously or under the influence of certain excitations, paroxysmal phenomena, during which the lower extremities are stiffened in extension, whilst, at the same time, they are drawn together, and, as it were, adhere to each other. These fits, which last for some hours, and occasionally for some days, are at first separated by intervals of greater or less length. Later on they become closer, and, at a given moment, permanent contracture is definitely established. When matters have reached this point, the following symptoms are observed—the lower extremities, as happened during the fits, are in extension; the thighs are extended on the pelvis, the legs on the thighs; the feet assume the attitude presented in talipes equinus (varus); the knees, moreover, are so closely drawn together that you cannot separate them without great effort. Both lower limbs are very generally affected simultaneously, and to the same extent; their rigidity is sometimes so marked that, in lifting one of them, whilst the patient is in bed, you, at the same time, lift the lower half of the body, all in one piece, as it were. Only in rare cases, and in the later stages of the diseases, does flexion of the thigh and leg predominate over extension.

Permanent contracture may invade, in exceptional cases however, the upper extremities, which are also generally placed in forced extension and straitly applied to each side of the body. We have here, gentlemen, to deal with a spasm which occupies simultaneously and with almost equal strength the antagonistic muscles, for, when the limbs are flexed, it is almost as difficult to extend them as it is to bend them when they are extended.

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[1] Erbstein ('Deutsches Archiv für Klinische Medicin,' t. x, fasc. 6, p. 595) has related the history of a patient who succumbed to disseminated sclerosis (the bulbo-spinal form), in whom, during life, *atrophy* of the anterior portion of the *tongue* had been observed. An histological examination afterwards showed—1st, numerous foci of degeneration, not only interposed between the fasciculi of the hypoglossal nerve at its origin, but also involving them and consequently interrupting their continuity. A section showed that the nucleus of the hypoglossal nerve was replaced by an islet of sclerosed tissue. 2d. The muscular fibres of the anterior portion of the tongue had undergone fatty degeneration; the lesion had invaded some of the muscular fasciculi at the base of the organ.

In a patient named Vincent, who succumbed to disseminated sclerosis, M. Charcot noticed atrophy of the muscles of the thenar eminence. The palm of the hand was hollowed out, and the tendons of the flexor muscles were very plainly defined. (B.)

When the extremity of one of the feet is grasped by the hand, and somewhat abruptly extended on the leg, there ensues almost immediately throughout the whole extent of the corresponding limb a sort of convulsive trembling, which recalls the tremulation determined by strychnine poisoning. This tremulation, which must not be confounded with the peculiar shake that supervenes on purposed movements, is not always limited to the limb in question; it is sometimes propagated to the other limb, and then the agitation may occasionally become so intense as to shake the whole body, and even the bed on which the patient reclines. It persists in some cases for several minutes, and even much longer, after cessation of the act which set it going. You may cause it to stop at once, as M. Brown-Séquard has shown, and as I have often since observed, by grasping, with the hand, one of the great toes of the patient and flexing it suddenly and forcibly. Immediately after this operation the tetanic rigidity and convulsive trembling cease in both members, which become temporarily "perfectly supple and pliable as after death, before rigor mortis supervenes."[1] The convulsive tremulation may be determined by faradization, by pinching the skin of the leg, or, more rarely, by kneading the limb, by the influence of cold, or by tickling the sole of the foot. It also comes on sometimes spontaneously, or at least apparently so, sometimes because of an effort made by the patient, as in vomiting, defecation, raising himself in bed, or getting out and placing his foot upon the floor. It is also provoked by an attempt to walk, for permanent rigidity does not always absolutely prohibit this act; the patients can sometimes hobble along on their toes, the heel being raised from the floor. Finally, this tremulation may also be temporarily produced, along with rigidity, even during the course of the paretic period, under the influence of one or other of the several modes of excitation which we have just reviewed.

Gentlemen, the phenomenon, whose principal characters I have here sketched, is nothing other than the *spinal epilepsy* described by M. Brown-Séquard. We observe it present in the case of Mademoiselle V— in what I have proposed to call the *tonic* form. This form, which is the type most commonly met with in gray multilocular induration, may be placed in opposition to the *saltatory* form, which predominates, on the contrary, in progressive locomotor ataxia and in some other spinal affections.

Permanent contracture of the limbs and spinal epilepsy must not any longer detain us. These symptoms, in fact, do not exclusively belong to multilocular sclerosis of the nervous centres. Far from it. They shall, therefore, be studied apart, both generally and in their relations with the different affections of the spinal cord in which they show themselves.

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[1] Brown-Séquard, 'Archives de Physiologie,' t. i, p. 158.

LECTURE VIII.

APOPLECTIFORM SEIZURES IN DISSEMINATED SCLEROSIS. PERIODS AND FORMS. PATHOLOGICAL PHYSIOLOGY. ETIOLOGY. TREATMENT.

SUMMARY.—Apoplectiform seizures. Their frequency in disseminated sclerosis. General considerations on apoplectiform attacks in general paralysis, and in cases of circumscribed cerebral lesions of old standing (hemorrhage and ramollissement). Pathogeny of apoplectiform seizures; insufficiency of the congestion theory. Symptoms: state of the pulse; elevation of the central temperature. Apoplectiform seizures in old cases of hemiplegia. Importance of temperature in diagnosis.

Periods in disseminated sclerosis. First, second, and third periods. Symptoms of bulbar paralysis. Forms and duration of disseminated sclerosis.

Pathological physiology: relation between symptoms and lesions.

Etiology. Influence of sex and age. Hereditary predisposition. Previous nervous affections. Occasional causes: prolonged action of moist cold; traumatism; moral causes.

Prognosis. Treatment.

GENTLEMEN: I purpose calling your attention to-day, in the first place, to certain cerebral accidents which may happen to complicate the symptomatology of cerebro-spinal disseminated sclerosis. I refer to *apoplectiform seizures*, which are occasionally encountered several times in the course of the disease, and which sometimes close the fatal scene. These attacks have not hitherto appeared in the case of Mademoiselle V—, whose clinical record is otherwise so complete in most respects; but nothing assures us that they will not some day show themselves. In fact, this is not a rare complication; I find it mentioned in about a fifth of the cases which I have collected, and I have personally observed it in at least three instances.[1]

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[1] Case III of the memoir of M. Vulpian, communicated by M. Charcot. Case of the patient Byr (Charcot); case of Nicolas, presented to the Société de Biologie, by M. Joffroy.

The group of symptoms, which constitutes an apoplectiform seizure, does not exclusively belong to multilocular sclerosis. It is found in a number of affections which involve several points of the cerebro-spinal axis at once, and particularly in progressive general paralysis. It is, indeed, in the latter disease that these *congestive attacks*—as they are commonly called, at least in France—have been specially studied on account of their frequency. They are met with there in all the various forms which they assume. The description of such attacks, in progressive general paralysis, has given rise to numerous divisions and subdivisions. But, in point of fact, all the varieties of form which clinical observation has revealed—I mean the graver kinds—may be classed as belonging to two fundamental types, namely:—

1st. *Apoplectiform attacks* (the "pseudo-apoplexy" of British authors), and

2d. *Epileptiform*, or *convulsive attacks*.

The characteristics of both types may, however, be intermingled and confounded in the same paroxysm. The first type only has been, up to the present, met with in disseminated sclerosis; but it cannot be doubted that, when observations relating to this disease shall have accumulated, they will enable us to fill up the picture.

Among the other organic diseases of the nervous centres in which apoplectiform or epileptiform attacks are frequently observed, I shall confine myself to certain circumscribed cerebral lesions of old standing, and accompanied by permanent hemiplegia. Such are *cerebral hemorrhage* and *brain-softening* when occupying regions of the encephalon, the lesion of which has the effect of almost certainly determining the cerebro-spinal alterations known under the name of *descending fasciculated scleroses*.

Between these partial lesions of the brain and progressive general paralysis it seems, at first glance, that no point of contact exists. However, gentlemen, here is a character which brings them together: the observations of M. Magnan and those of Herr Westphal have shown that, in general paralysis, there is very often superadded to the lesions of periencephalitis a sclerous alteration, sometimes diffuse and sometimes fasciculated, which occupies the crura-cerebri, pons Varolii, medulla oblongata, and certain regions of the spinal cord, at the same time. Now, these cerebro-spinal lesions (as much on account of their mode of distribution as because of the peculiar nature of the morbid process) deserve to be assimilated to the descending fasciculated scleroses consecutive on hemorrhage or softening of the brain. We know, on the other hand, that, in multilocular sclerosis, the sclerosed patches occupy not only the spinal cord and the brain proper, but are likewise very commonly found in different parts of the isthmus cerebri, and particularly in the bulbous rachidicus. You see, by this, that the existence of irritative lesions, disseminated nearly everywhere in the cerebro-spinal axis, but always present in the isthmus cerebri, is a character common to all those affections, so different in appearance, in which the so-called *congestive* attacks supervene. I would especially point out to your attention the constant existence of the bulbar lesion, which is, in all probability, a predominant element in the production of these attacks.

However this may be, gentlemen, we have here permanent alterations of slowly progressive evolution. They cannot, consequently, without the assistance of other lesions, explain the development of accidents which are, for the most part, suddenly produced, and which may rapidly disappear without leaving any trace. I am not unaware that many physicians, even at the present day, put forward the theory of a partial sanguine congestion—a fluxion which, according to the needs of the case, should affect this or that portion of the encephalon. As regards myself, I cannot endorse this hypothesis. In order to justify my scepticism in this matter, I will appeal to the reminiscences of those among you who, in this hospital, were attached to the department for the insane. How many times have they not been disappointed in not finding, on post-mortem examination, the congestive lesion, which they expected? But I shall appeal, above all, to the cases which I have had opportunities of collecting in my accustomed field of study. Many a time have I had occasion to see patients, long suffering from hemiplegia, the result of brain-softening or intracephalic hemorrhage, succumb to epileptiform or to apoplectiform attacks. Now, in such cases, no matter what attention I gave to the autopsy, I have ever found it impossible to discover, whether in the nervous centres or in the viscera, any recent congestive lesion, œdematous or other, which could explain the grave symptoms that had characterized the fatal termination of the disease. I have never met with any but old lesions—ochreous foci, yellow patches, or foci of cellular infiltration—on which depended the hemiplegia, and the secondary degenerations of the mesocephalon and of the cord, which are the consequences of these partial lesions of the cerebral hemispheres. In short, I believe that, in the present state of science, the absence of proper lesions is, anatomically speaking, a common characteristic of these attacks, whatever be the form they assume or the disease with which they are connected.

In what relates to the symptomatology of the apoplectiform and epileptiform attacks, in order not to enter upon the details of a regular description, I shall confine myself to mentioning the following peculiarities. The scene generally opens unexpectedly, without any marked preliminaries, sometimes by rapid and more or less intense obnubilation of the intellectual faculties, sometimes by profound coma, suddenly supervening. In certain cases convulsions are added, which recall those of ordinary epilepsy, but which are usually localized in one side of the body (*epileptiform attacks*). In other instances there are no convulsions (*apoplectiform attacks*). In both cases it is frequent to find, developed from the outset, a more or less complete hemiplegia, sometimes with flaccidity, sometimes, but more rarely, with rigidity of the paralyzed members. The symptoms may gradually grow worse in the course of a few days and induce death. This is usually heralded by the rapid development of eschars on the sacral region. If, on the other hand, the patient is destined to survive, the disappearance of the symptoms soon becomes manifest, hemiplegia is the only one that holds out for some time, but sooner or later it also dissipates without leaving any trace of its existence.

These attacks usually recur several times, generally after long intervals, during the course of the disease. So far as disseminated sclerosis is concerned, they have been noticed thrice in Case III of M. Vulpian's memoir, thrice in Zenker's case,[1] and up to seven times in that recorded by M. Léo[2]. In every instance, these fits left after them a notable and persistent aggravation of all the symptoms of the original disease.

The sketch which I have given you, gentlemen, would be too imperfect if I did not call your attention to the troubles of circulation and temperature which, as a general rule, show themselves in the course of these attacks. The *pulse* is always more or less accelerated; but, besides (and this is the important point), the *temperature* of the central parts rises rapidly; it may, in the hours immediately following the invasion, reach 38.5° C. (= 101.3° F.), or even 39° (= 102.2° F.), and frequently, at the end of twelve or twenty-four hours, it rises to 40° (= 104° F.), and remains at this elevation for some hours, without necessarily entailing a fatal result. But if the patient is to survive, the temperature soon diminishes rapidly. An increase above 40° C. is almost always followed by a fatal termination.

These modifications of central temperature have been studied by Herr Westphal in the epileptiform and apoplectiform attacks of *progressive general paralysis;* I have met with them again in the attacks which supervene in patients suffering from *hemiplegia of old standing*, consecutive on *hemorrhage* or on *softening of the brain.* In order the better to settle your ideas upon this subject I think it will be useful to summarize the details of two cases relating to the last-named species.

The first case is that of a woman, aged thirty-two years, affected by hemiplegia of the right side, dating from childhood. There existed general atrophy, rigidity, with shortening of the limbs, and paralysis, such as are generally found in like cases. This woman was subject to epileptiform attacks. She was brought to the infirmary some hours after a more than usually severe attack. On the evening of her admission her temperature was above 38° C. (=100.4° F.); next day it had reached 4° C. (=104° F.). The fits became subintrant; they were repeated about a hundred times a day. Eschars formed rapidly on the sacral region, and death supervened the sixth day. On that day the rectal temperature stood at 42.4° C. (=108.32° F.). On post-mortem examination there was found, at the surface of the left cerebral hemisphere, a considerable depression answering to a yellow patch, the remnant of a vast focus of ramollissement. The whole hemisphere, moreover, was atrophied. No trace of a recent lesion could be found, neither in the nervous centres nor in the viscera.

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[1] Bourneville et Guérard, *loc. cit.*, p. 112.

[2] *Ibid.*, p. 112.

The second case is that of a woman, aged sixty years, afflicted with right hemiplegia consecutive on cerebral hemorrhage, dating from two years previously. This patient had already experienced several epileptiform or apoplectiform attacks, which, however, were generally slight. One day an intense and prolonged epileptiform attack supervened, which was followed by an apoplectiform condition. Two hours after the setting in of these accidents, the rectal temperature was 38.8° C. (=101.84° F.); five hours later, it rose to 40° C. (=104° F.). Next day, in spite of the cessation of convulsions, the temperature was 41° (=105.8° F.); and the day following, being the day of her death, it reached 42.5 C. (=108.5° F.). The autopsy showed two ochreous foci, one occupying the corpus striatum, the other the substance of a convolution. There existed no recent lesion capable of explaining the accidents which had determined death.

I have as yet had no opportunity of following, day by day and at different periods of the day, the changes of central temperature in a case of *apoplectiform seizure* supervening in a patient affected with *disseminated sclerosis*. Nevertheless, we can gather partial results from different cases, which leave no doubt that, even in this respect, matters proceed exactly in the same way in multilocular sclerosis, as in progressive general paralysis and in circumscribed lesions of the cerebral hemispheres. Thus, the patient whose history has been related by Herr Zenker was, towards the close of his life, taken with an apoplectiform attack, followed by hemiplegia of the right side. Now, on the day of the seizure, his pulse being at 136, the temperature reached 39.6° C. (=103.28° F.). Next day, the thermometer marked 40° C. (=104° F.). The day after, the paralysis had ameliorated and the temperature had fallen back to the physiological figure. In the case of the patient Nolle, narrated by M. Léo, an apoplectiform attack came on in the evening. Next morning early, the pulse numbered 144, and the temperature stood at 38.5° C. (=101.3° F.). This attack, the seventh that the patient had experienced, was followed on the same night by death. In the case of N—, whose record was compiled in my wards by M. Joffroy, five hours merely after the invasion of an apoplectiform attack, with incomplete loss of consciousness and general resolution of the members, the rectal temperature stood at 40.3° C. (=104.54° F.), and the pulse at 120. Next day the apoplectiform symptoms were dissipated, and at the same time the pulse and the temperature had returned to what they were in the normal state.[1]

If I have dwelt with some tenacity on the changes which the temperature of the body presents, in the apoplectiform and epileptiform seizures of general paralysis, and of some other cerebro-spinal affections, it is because, in my judgment, we find a characteristic therein which may, in certain cases, be profitably used in diagnosis. It is not necessary, I think, to enter into a long discussion in order to show how difficult it is, in presence of a patient who has just been stricken with apoplexy, accompanied or not by convulsions, to decide from the mere contemplation of external symptoms whether we have to deal with *true apoplexy*, resulting from the actual formation of a focus of cerebral hemorrhage or of ramollissement, or whether, on the contrary, we have before us a simple *congestive attack*. Well, an examination of the central temperature would supply, in such cases a decisive test. I have, in fact, demonstrated by repeated observations[2] that in true apoplexy, especially when it depends upon cerebral hemorrhage, the temperature constantly diminishes, some moments after the attack, and afterwards remains, generally for at least twenty-four hours, below the normal standard, even when intense and reiterated convulsive fits occur. Now, we have just seen that, in the so-called congestive attacks, the temperature, on the contrary, from the invasion of the first symptoms, rises above the physiological standard and tends to become gradually more and more elevated during the whole continuance of the attack.

PERIODS AND FORMS OF DISSEMINATED SCLEROSIS.

Gentlemen, after having considered, one by one, the different elements which compose the symptomatology of multilocular sclerosis where we have to deal with a complete case, one which has already arrived at an advanced stage of its evolution,—it is next proper to show, in a general view, how these elements are grouped and arranged in the different phases and forms of the disease. The affection is, in truth, far from presenting itself clothed in all its attributes, at every epoch of its course. At the outset it may be constituted by the union of two or three symptoms only; and, besides, there are cases where the symptomatic series remains incomplete until the fatal end. Now, it is, especially, when the disease is yet at an early stage, or when it assumes an imperfect form, that it is important to know how to recognize it by the slightest indicia.

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[1] 'Société de Biologie,' t. i, 5 série, 1869-70, p. 145.

[2] Charcot, "Note sur la température des parties centrales dans l'apoplexie liée à l'hémorrhagie cérébrale et au ramollissement du cerveau," in 'Comptes Rendus des Séances de la Société de Biologie,' t. iv, 4e série, 1867, p. 92. See also Charcot, 'Leçons sur la thermométrie clinique, publiées dans la *Gazette hebdomadaire*,' 1869, pp. 324, 742, 821; Bourneville, 'Études cliniques et thermométriques sur les Maladies du Système Nerveux,' Paris, 1870–73.

I have proposed to establish three periods in the progressive development of the disease. The first extends from the moment when the first symptoms appear to the epoch when the spasmodic rigidity of the members reduces the patient to almost absolute impotence. The second comprises the space, usually of considerable length, during which the patient, confined to bed, or barely able to take a few steps about the room, still preserves the integrity of his organic functions. The third commences at the moment when, all the symptoms of the disease becoming simultaneously aggravated, the functions of nutrition suffer in a manifest manner. We will take occasion, as regards this ultimate period, to notice the disorders which, in the common order of things, mark the last phase of the disease and accelerate its fatal termination.

I.

First period.—The mode of invasion and of concatenation of symptoms presents certain varieties which deserve to be pointed out to your notice.

Sometimes, the drama is begun by the cephalic symptoms. Thus, the patients commence by complaining of habitual giddiness, and more or less transient diplopia; little by little, difficulty of enunciation, and finally nystagmus, show themselves. The union of these symptoms would already constitute a sufficiently characteristic group, one which, even if tremor provoked by movement and paresis of the limbs were not superadded, should of itself enable us to establish a diagnosis, on strong probabilities.

But such is not the most common mode of invasion. Generally, the spinal phenomena first reveal themselves, and so common is this circumstance that during many months—nay, even for years—the patients may present no symptoms other than an enfeeblement, a more or less marked paresis of the lower extremities, displaying a tendency to become aggravated, in a slowly progressive manner, and to extend to the upper extremities. In such a case, the position of the clinical observer is necessarily an extremely difficult one. For, in short, paresis of the lower limbs is a somewhat trite symptom, one common to a crowd of different diseases; still, it shows itself in multilocular sclerosis, as you remember, with some peculiar features which may indicate the right path to follow. Thus, however marked it may be—setting aside exceptional cases where the lesion predominates in the posterior columns—it is not accompanied by any trouble of sensibility, nor by any perceptible disorder of nutrition in the muscular masses. Add to this that, as a rule, there is no functional derangement of bladder or rectum. Finally, it is not rare to meet with *remissions*, and even with complete *intermissions*, which give rise to hopes of a decided cure.[1] But it is clear that these indicia, even with the aid of all the others, only supply very vague data. Certainty can hardly be secured unless the peculiar tremor, or some of the cephalic symptoms, are superadded to the spinal symptoms.

Hitherto, gentlemen, I have shown you the invasion and ulterior concatenation of accidents as slow and progressing in a uniform manner. That, in fact, is by far the most usual case; but it is important you should know that, in certain exceptional circumstances, the disease may set in suddenly and unexpectedly, or after a few preliminary symptoms, of little significance.

Thus, vertigo and diplopia having suddenly shown themselves, paresis and titubation may follow in a few days, so that the disease is thus, as it were, immediately established. This, to take one case amongst several, is what happened as regards the young woman named Vinch—, whom some of you may have seen in our wards.

Sometimes the beginning is marked, as in the case of one of Valentiner's patients, by an abrupt invasion of paresis in one of the lower extremities; or again, as occurred in M. Léo's case and in that of one of my patients, whose history M. Vulpian has related,[2] the invasion is inaugurated by an apoplectiform attack, preceded for some days or weeks, by vertigo and cephalalgia, and followed by temporary hemiplegia.

Finally, gentlemen, there is yet another variety, to which I must call your attention, where the invasion is marked by an affection which is mostly regarded as foreign to the principal disease, although it is, in my opinion, intimately bound up with it, on the contrary, by a link not recognized until now. I allude to the *gastric* or *gastralgic crises*, whichever you please to call them, that are occasionally very severe, and are accompanied by lypothymia, by repeated vomiting, &c. These crises have often opened the drama, and been quickly followed by the usual symptoms of multilocular sclerosis; it is not rare, also, to find them several times recurring and intermingling with these symptoms, during the early stage of the disease. Of this class, a case reported by M. Liouville[1] and that related by Herr Zenke furnish good examples. These accidents are all the more worthy of notice inasmuch as we shall find them again, with nearly the same characters, in other forms of sclerosis of the spinal cord, and particularly in fasciculated posterior sclerosis (*locomotor ataxia*), but chiefly in its initial phases. In such a case, these gastric crises, coinciding or alternating with the fulgurant pains of the limbs, may actually be, along with diplopia and perhaps a little titubation when the eyes are closed, the only symptoms of the disease in question, whose true nature is then too often misunderstood.[2] These same gastric crises are found, as my friend Dr. Duchenne (de Boulogne) and I have observed, in the form of *subacute* or *chronic central myelitis*, which reproduces the symptoms of *general spinal paralysis*. But I do not wish to delay any longer on this subject, which I intend soon to resume and to discuss in detail, as its importance deserves.

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[1] In our memoir, we summarized a certain number of cases in which remissions were found so complete as to enable the patients, who had been paralyzed, to resume their occupations. (See *loc. cit.* obs. iv, ix, x, xi, &c.) In an observation recorded by M. Vulpian, which we also quoted (p. 139), there was a series of alternate ameliorations and aggravations. We shall briefly indicate them :

When the disease was still recent, there supervened, after an attack of smallpox, a quasi-complete recovery. This improvement lasted for three years. At the end of that time, the menses were suppressed ; new, but slight, symptoms showed themselves, which disappeared on restoration of the catamenia. Two years after, the patient had an attack of jaundice, followed by new symptoms. These improved, but on bronchitis supervening, the paresis of the limbs reappeared in a more marked form, and, after successive remissions and recrudescences, became permanent.

Sometimes the remission is incomplete, and only affects certain symptoms, particularly incontinence of urine and of feces. In a patient, whose case was noted by Herr Baerwinkel, there was also a brief remission. (B.)

[2] Vulpian, "Note sur la Sclerose en Plaques de la Moelle Épinière," Obs. ii, 'Mémoires de la Société Médicale des Hopitaux,' 1869.

II.

Second period.—In general, from the close of the first period, multilocular sclerosis shows itself arrayed in most of the symptoms which characterize it. These symptoms become aggravated and intensified during the second period, and spasmodic contraction of the limbs is superadded, either with or without the accompaniment of spinal epilepsy, in consequence of which the patients who, until then, had been able to walk or hobble, with more or less difficulty, are thenceforth rendered almost quite powerless, and definitely confined to their rooms or beds. The contracture which marks the commencement of this period is almost always a very tardy symptom; it seldom shows itself till two, four, or even six years after the appearance of the first accidents of multilocular sclerosis.

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[1] 'Mémoires de la Société de Biologie,' 5e série, t. i, p. 107, Paris, 1870.

[2] See what M. Charcot has said, in reference to this subject, in his lectures delivered at La Salpêtrière in 1868 (Dubois, 'Etude sur quelques points de l'ataxie locomotrice,' Paris, 1868, "Des crises gastriques," p. 56; 'Leçons sur les anomalies de l'ataxie locomotrice,' 1873, leçon ii, p. 32).

III.

Third period.—The commencement of this final period is marked, as I mentioned to you, by the progressive enfeeblement of the organic functions; inappetency becomes habitual, diarrhœa frequent, and soon a general emaciation supervenes which grows more and more evident.[1] At the same time, there ensues an aggravation of all the symptoms proper to this disease, the obnubilation of the intellect proceeds even to dementia, the difficulty of enunciation is carried to its extreme, and the patient can only utter an unintelligible grunting; then the sphincters become paralyzed, and it is not rare to find the mucous coat of the bladder affected with ulcerous inflammation. Then, on the sacral region and on all points of the lower limbs submitted to prolonged pressure, eschars appear which occasionally assume enormous dimensions, and, consecutively, comes the whole series of accidents which depend on this complication, purulent burrowing sores (*fusées*), purulent or putrid poisoning, etc. Death follows without delay.

In most cases the patient's existence may be abridged by some intercurrent disease;[2] pneumonia, caseous phthisis, and dysentery may be numbered amongst the most frequent of these terminal affections.[3]

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[1] At this period of the disease, especially, we notice the supervention of disorders which may, perhaps, be classed among trophic troubles. Such are—1st, softening of the vertebræ, of the trochanters, of the head of the tibia, of the bones of the tarsus, &c. (Bourneville et Guérard, *loc. cit.*, cas du Docteur Pennock, p. 83); 2d, a cyphosis and (right) scoliosis, mentioned in one of Friedreich's cases (B. et G., *loc. cit.*, pp. 213 and 214); 3d, an effusion of liquid into the two femoro-tibial articulations (Obs. de M. Malherbe). (B.)

[2] In the cases which have since been published, we most usually find the terminal diseases indicated by M. Charcot. It follows from the statistics we have collected that pulmonary diseases (pneumonia, purulent pleurisy, tubercular phthisis) are by far the most prominent. We should also mention the occurrence of *acute bed-sore*, of *pyelo-cystitis* (one case), and of *œdema glottidis* (one case). (B.)

[3] In this manner, the patient Vauthier (the subject of the preceding lecture) succumbed, and the patient Bezot, who long occupied bed No. 10, Salle St. Luc. We shall rapidly summarize the principal facts of their clinical history:—

I. Vauth—(Josephine C.) was admitted March 21st, 1867, to M. Vulpian's wards, and died January 7th, 1871 (aged thirty-two), in M. Charcot's charge. From fourteen to twenty-one years of age, she suffered from vertigo followed by vomiting. Pregnancy, at twenty-one, put an end to vomiting. Disseminated sclerosis showed itself at the age of twenty-three years, six months: weakness of the lumbar region, very great fatigue of the lower limbs, lancinating pain in the right leg, enfeeblement of the sight, diplopia. At twenty-five years, feebleness of the arms, which are occasionally affected by pains.

1867.—Nystagmus, diplopia. Integrity of the muscular masses, loss of idea of position as regards lower limbs. Paresis and tremor of the upper extremities. Tactual sensibility largely lost everywhere. Momentary improvement under nitrate of silver.

1868.—The patient can no longer stand erect; the symptoms are more marked on the right side than on the left; the tremor of the upper extremities has augmented. Frequent fulgurant pains, especially in the left half of the face. Fits of giddiness coming on at close intervals. Nystagmus more marked. In May, I have reserved for special mention the appearance of some symptoms of *bulbar paralysis*, because they may, by an abrupt aggra-

M. Vulpian administered two pills of 0.025 gram. (or nearly ⅓ grain) of extract of Calabar Bean. Soon after, a fit of weakness, tremor exaggerated, cold sweats, pallor of the face (these phenomena are, perhaps, due to the Calabar Bean). From July, three pills of Calabar Bean. In November, M. Vulpian suppresses the Calabar Bean, and as incontinence of urine has latterly supervened, he prescribes three pills of 0.03 gram. (or nearly ½ grain) of extract of Belladonna. The incontinence of urine, after presenting some transient improvements, ceased altogether in the course of December.

1870 (January).—Psychic disorders (see *ante*, p. 160). In the course of this year the symptoms noted augmented in severity; and, besides, symptoms of bulbar paralysis made their appearance. These became rather rapidly worse, and the patient died, as it were asphyxiated, Feb. 7th, 1871.

Autopsy.—Numerous sclerosed patches found to exist in the brain and spinal cord. On account of the *ataxic symptoms* presented by the patient, the lesions of the spinal axis deserve mention. There were sclerosed patches throughout the whole length of the lateral columns. As to the *posterior columns*, they are affected nearly throughout, but, principally, from the lower extremity of the dorsal region upwards. Fig. 15 represents the lesions observed on a section taken from the upper part of the lumbar region. At this level the posterior columns are completely invaded (fig. 15, *c*), but especially affected in the mid-region. The lateral columns are comparatively less injured.

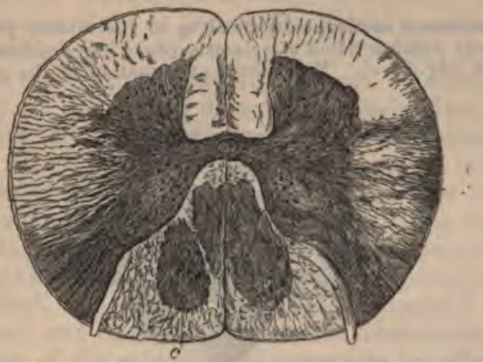

Fig. 15.—Representation of the lesions observed on a section taken from the uppermost portion of the lumbar region. The posterior columns are invaded throughout their breadth, and the lesion predominates in their middle region.

II. Bez—(Pauline). aged thirty-five, child's nurse, admitted Feb. 17th into M. Charcot's wards. To the ordinary symptoms of disseminated sclerosis were added, about the month of May, dyspnœa and dysphagia. The difficulty of deglutition compelled the patient to eat very slowly. Return of food, through the nasal orifices, was not observed until near the end. The patient died of asphyxia, June 12th, without any râles having been noticed in the lungs.

Autopsy.—Sclerosed patch on the chiasma of the optic nerves, invading the tractus opticus. Sclerosed patch in the ventricles and in the centrum ovale. In a section made a centimétre above the inferior border of the protuberantia annularis, on a level with the apparent origin of the trifacial nerve, a large and irregular patch of sclerosis is found (fig. 16, *b′ b′*).

vation, precipitate the course of events, and induce the fatal termination, even before the manifestation of the phenomena of the final period. Contemporaneously with increased difficulty of utterance, there appears a difficulty of deglutition which, though transient at first, soon becomes permanent. Then, from time to time, paroxysms of dyspnœa show themselves, of less or more gravity, and death may supervene during one of these fits. I have recently observed two cases which terminated in this manner. On a post-mortem examination it was seen, in both these cases, that a patch of sclerosis had invaded the floor of the fourth ventricle, where it enveloped the originating nuclei of most of the bulbar nerves. (See foot-note 3, p. 176.)

After the details which I have laid before you, it seems useless to undertake the particular description of the different *forms* which multilocular sclerosis may assume. The *cerebral* and *spinal* forms correspond to an incomplete invasion of the nervous centres by the sclerosis; it is if you like, the disease arrested in its development, in its progress either of ascent or of descent. The symptomatic series is, therefore, as it were, curtailed; but the symptoms, considered singly, are not modified. The first, or cerebral, form is very rare; the second, or spinal, is, on the contrary, very frequent; but

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Another transverse section, corresponding to the middle part of the corpora olivaria, reveals another patch of sclerosis (fig. 17 *c*) apparently involving the pneumogastric (fig. 17 *a*). Microscopic examination of the nerves showed the existence of numerous fatty degenerated tubes in the hypoglossal nerve, and traces of irritation in Schwann's sheath in the pneumogastric nerve. As to the other organs, and particularly the pharynx, the larynx, and the lungs, they were all healthy.

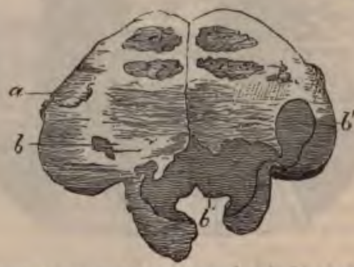

Fig. 16.—*a*, Pneumogastric; *b*, small sclerosed patch; *b'*, large sclerosed patch.

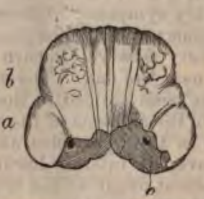

Fig. 17.—*a*, Pneumogastric nerve; *b*, hypoglossal nerve; *c*, sclerosed patch.

on the whole the *cerebro-spinal* form constitutes the normal type, that which we most often meet with in practice.

Cerebro-spinal multilocular sclerosis completes, generally speaking, its whole evolution in the space of from six to eight years;[1] this establishes another contrast between it and paralysis agitans, the normal duration of which is much longer. The spinal form usually gives the most lengthy respite; it may not terminate its course for a space of twenty years, or even longer still.[2]

PATHOLOGICAL PHYSIOLOGY; ETIOLOGY; PROGNOSIS, AND TREATMENT.

In order to conclude this study, gentlemen, it remains for me to discuss the pathological physiology, the etiology, and finally the therapeutical treatment of multilocular sclerosis of the nervous centres. Unfortunately, the facts and documents which refer to these different subjects are few in number, and as yet mostly imperfect, so that I shall be compelled to confine myself to a few summary observations.

A. The cause of the very singular mode of arrangement under which the sclerosed islets are distributed in different parts of the central nervous system, is, at present, completely unknown to us. Herr Rindfleisch[3] has stated that the starting-point for the formation of sclerosed foci resides in the vascular system. According to him, the inflammation of the walls of the smaller vessels, always to be met with in the centre of foci during the process of formation, would be the initial fact; from this central point the irritation is propagated to the reticulum of the neuroglia, whence it radiates in all directions. It is evident, however, that this explanation only sets the difficulty a little farther back. Besides, this predominant part accorded to vessels in the evolution of the morbid process is anything but demonstrated. I am even very much disposed to believe, judging from my own observations, that the alterations of vascular tissue and those of the reticulum advance side by side in parallel lines, without acting reciprocally on each other. Be this as it may, the question arises whether, if the position of the sclerosed islets, in the different portions of the nervous system, be given, we can deduce therefrom the production of phenomena which, in their totality, constitute the symptomatology of disseminated sclerosis? This is, at least to some extent, possible. We have already shown you that the motor inco-ordination, the loss of sense of position and the fulgurant pains which are found in a certain number of cases, may, in such cases, be attributed to the sclerous invasion of the posterior columns of the cord for a certain altitude. On the other hand, the customary predominance of sclerosed patches along the course of the antero-lateral columns accounts, as I shall soon demonstrate to you, for the almost constant existence of the paresis or of the paralysis of the limbs, which is sooner or later followed by permanent contracture. The nystagmus and the difficulty of enunciation are correlated with the habitual localization of nodules in the substance of the protuberantia annularis and the bulbus rachidicus. But there are a large number of other symptoms, the interpretation of which presents much greater difficulty. Such, for instance, is the peculiar tremor which is manifested during certain attitudes of the body, and in the execution of intentional movements. I have expressed the opinion that the long persistence of the axis-cylinders, deprived of medullary sheathing, in the midst of the foci of sclerosis, probably plays an important part here. The transmission of voluntary impulses would still proceed by means of the denuded axis-cylinders, but it would be carried on irregularly, in a broken or jerky manner, and would thus produce the oscillations which disturb the due execution of voluntary movements.

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[1] It is rather difficult, at present, to ascertain the mean duration of disseminated sclerosis. In a first collection (Bourneville et Guérard, *loc. cit.*, p. 148), comprising seventeen cases, we found a mean of from eight to ten years. In another, including thirteen new cases, we found a mean of seven and a half years. The minimum of the duration of the disease was one year (case of M. Malherbe, in 'Journal de Médecine de l'Ouest,' 1870, p. 168, and Buschwald, "Ueber Multiple Sklerose des Hirns und Ruckenmarks," in 'Deutsches Archiv für Klin. Medicin,' c. x, fas. iv und v, p. 478, 1872). The maximum is from sixteen to seventeen years. (B.)

[2] In three cases of disseminated sclerosis, with predominance of lesions of the posterior columns, the disease lasted fourteen, twenty-one, and twenty-eight years. (Bourneville, 'Nouvelle étude sur quelques points de la sclérose en plaques disséminées,' 1869.)

[3] Rindfleisch, "Histol. Detail zu der Grauen Degeneration von Gehirn und Ruckenmarks" ('Virchow's Archiv,' 1863, t. xxvi, p. 474).

This resistance of the axis-cylinders is certainly not a phenomenon exclusively pertaining to multilocular induration; but it is here manifested in a more marked manner than in other forms of sclerosis of the nervous centres. It may also be quoted, I think, to account for the slowness with which the paretic symptoms advance in disseminated sclerosis, and for the long space of time which elapses before they give place to complete paralysis and permanent contracture.

B. What is known in reference to the conditions that preside over the development of disseminated sclerosis comes to very little. It seems, however, to be established at present, that the disease is far more common in females than in males. Thus, of all the instances which I collected in my first treatise, only three or four related to men. The cases which have since been published have not modified this result in any perceptible manner. Adding to the eighteen cases which are mentioned in the monograph of MM. Bourneville and Guérard, sixteen new cases, we get a total of thirty-four cases, of which nine relate to males, and twenty-five to females.

Judging from the same records, it follows that this is a disease of youth, or of the first half of adult age. It has been observed in patients aged fourteen, fifteen, and seventeen years,[1] but it seems most frequently to set in between twenty-five and thirty years. It rarely makes its appearance after thirty. On the other hand, forty years seem to be the ultimate limit of life for patients affected by disseminated sclerosis.

With respect to the influence of hereditary predisposition, we have only one case to mention in which it seems to have played a certain part. This example has been communicated to us by Dr. Duchenne (de Boulogne).

In the pathological antecedents of the patients themselves we generally find nothing but vague indicia. Hysteria was present in some cases, but in most we only find mention made of ill-determined neuropathies, occasional hemicrania, or neuralgias.[2]

Amongst *occasional causes*, we frequently find mention made of the prolonged action of moist cold.[3] In one case, the first symptoms are alleged to have appeared a short time after a fall.

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[1] In a work by M. Leubé ("Ueber multiple inselformig Sklerose der Gehirns und Ruckenmarks," in 'Deutsches Archiv,' 8 Bd., 1 heft, 1870, p. 14) we find the record of the case of a child who presented the first symptoms of disseminated sclerosis at the age of seven years. She died aged fourteen years and six months. Summary: slight nystagmus; right facial paralysis; marked ataxia of the extremities, especially on the left side; tremor of the head; difficulty of utterance; atrophy of the legs. *Autopsy*: sclerosis of the pons Varolii and annexes, almost general on the right, disseminated on the left. The cerebrum and cerebellum present, in their cortical layers, a double degeneration, whitish-yellow, or steel-gray, partly diffused, partly disseminated in patches. In the cord, and principally in the medulla oblongata, the sclerosis occupies, firstly, the posterior columns, next the lateral columns, and finally the anterior columns. (B.)

[2] There is, however, an etiological cause which deserves mention, namely, the influence of certain acute diseases on the development of sclerosis. The following facts are given in support of this assertion.

1st. In a case given by Erbstein ('Deutsches Archiv für Klinische Medicin,' t. x, fasc. 6, p. 596) disseminated sclerosis set in during convalesence from typhoid fever. The patient, then, suffered from debility of the lower extremities, and a difficulty of enunciation; the words were scanned, and the pronunciation was somewhat indistinct and monotonous.

2d. A patient in M. Charcot's charge, Nic—(Julie) noticed a certain degree of weakness in her lower limbs, on recovering from an attack of cholera. Some short time after, she had an attack of typhoid fever, after which the feebleness of her limbs augmented in a slow but continuous manner, to such an extent that she was soon obliged to use a cane. (A. Joffroy, 'Mémoires de la Société de Biologie,' 1869, p. 146.)

3d. In the case, recorded by MM. Fontaine and Liouville, it is mentioned that the first signs of sclerosis were preceded by copious bilious vomitings, which lasted from ten to fifteen days. (H. Liouville, in 'Mémoires de la Société de Biologie,' 1860, p. 107.)

4th. Finally, we will mention the case of a woman named Dr—(Hortense), in whom the first symptoms of disseminated sclerosis appeared soon after she had had a severe attack of smallpox. (B.)

[3] A patient, according to Herr Baerwinkle, experienced a difficulty in executing movements with the right leg, three days after having fallen into water. The action of moist cold has a reality in this case, because the patient allowed his wet clothes to dry upon him. (B.)

But the circumstances most commonly assigned as causes of this disease, by patients, appertain to the moral order—long-continued grief or vexation, such, for instance, as may arise from illicit pregnancy, or the disagreeable annoyances and carking cares which a more or less false social position entails. This is often the case as regards certain female teachers. Having said so much with respect to women,[1] the question of male sufferers arises. These are, for the most part, persons who have lost caste, and who, thrown out of the general current, and too impressionable, are ill-provided with the means of maintaining what, in Darwin's theory, is called the "struggle for life." In short, the etiology is a somewhat trite one, such as may be met with again, as it were, at the beginning of all the chronic diseases of the central nervous system.

C. The *prognosis* has hitherto been of the gloomiest. Shall it be always thus? It is to be hoped that, when the disease has become better known, the physician will learn how to take advantage of that spontaneous tendency to remission which has been noticed in a great number of cases. Nor must it be overlooked that, at the present time, the real nature of the disease is not recognized until the lesions have become well marked, and are consequently but little amenable to the influence of therapeutic agencies.

D. After what precedes, need I detain you long over the question of treatment? The time has not yet come when such a subject can be seriously considered. I can only tell you of some experiments which have been tried, the results of which, unfortunately, have not been very encouraging.

Chloride of gold and phosphate of zinc appear to have rather exasperated than improved the symptoms. Strychnine has sometimes caused cessation of the tremor, but its influence has always been transient. The same is to be said of nitrate of silver. In several cases, which I have noted, it seems to have had a very favorable influence over the tremor, and the paresis of the limbs, but this influence was not of long duration.

The exhibition of this drug is formally contra-indicated by the existence of permanent contracture, and especially of spinal epilepsy; its employment, in such cases, would almost certainly have the effect of exasperating these symptoms. The hydropathic treatment seems to have produced a temporary amendment in one case; in another, on the contrary, it completely failed.

Arsenic, belladonna, ergot of rye, and bromide of potassium have been likewise administered, in disseminated sclerosis, without any marked benefit. The same may be said of faradization and of galvanism. As regards the employment of the continued current, however, further experiments are required before we can form a definite judgment.[1]

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[1] The 'Lancet' (1873, vol. i, p. 236) has published the summary of a case of disseminated sclerosis recorded by Dr. Moxon, at Guy's Hospital, in which we find the following causes mentioned—*a*, febrile disease, accompanied by diarrhœa, which lasted for several weeks; *b*, a violent moral emotion experienced by the patient on seeing her husband in bed with another female. (B.)

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[1] Other drugs have also been employed, without better success than resulted from the use of those enumerated by M. Charcot. Such are, phosphorized oil, iodide of phosphetylamine, and Calabar Bean. Since the publication of the first edition of these Lectures several works or observations concerning disseminated sclerosis have appeared. As they only confirm the descriptions traced by M. Charcot, we confine ourselves to a simple catalogue. 1st. Timal, "Etude sur quelques complications de la sclérose en plaques disseminées." Thèse de Paris, 1873. 2d and 3d. H. Schule, "Beitrag zur multiplen Sklerose des Gehirns und Ruckenmarks," in 'Deutsches Archiv für Klinische Medicin,' 1870, Bd. vii, p. 259. "Weiterer Beitrag zur Hirn Ruckenmarks Sklerose" (ibid., 1871, Bd. viii, p. 223). Baldwin, "A Case of Diffused Cerebral Sclerosis" ('Journal of Mental Science,' 1873, July, p. 304). 5th. Moxon, "Two Cases of Insular Sclerosis of the Brain and Spinal Cord" ('The Lancet,' vol. i, p. 471, 609, 1875). 6th. Buzzard, "Disseminated Cerebro-Spinal Sclerosis," (ibid., vol. i, p. 45). 7th. Moxon, "Eight Cases of Insular Sclerosis of the Brain and Spinal Cord" ('Guy's Hospital Reports,' 3d series, t. xxi, London, 1875).

PART THIRD.

HYSTERIA.—HYSTERO-EPILEPSY.

LECTURE IX.

HYSTERICAL ISCHURIA.

SUMMARY.—Introduction. Hysterical ischuria. Differences which divide it from oliguria. General considerations. Supplementary vomiting. Historical Sketch. Causes which have thrown doubt on the existence of hysterical ischuria. Distinction between calculous ischuria and hysterical ischuria.

Case. Hysterical paralysis and contracture. Complete hemianæsthesia. Hemiopia and achromatopsia. Ovarian hyperæsthesia. Retention of urine. Tympanites. Convulsive seizures; trismus. Manifestation of hysterical ischuria. Precautions taken to guard against error. Complete anuria. Uræmic vomiting. Relation of the quantity of urine excreted to the vomited matter. Chemical analysis of vomited matter, urine and blood. Suspension of phenomena.

Re-appearance of hysterical ischuria. New results of chemical analyses.

Serious nature of common anuria and of experimental anuria. Limit of the duration of accidents compatible with life. Influence of the evacuation of even a minute quantity of urine. Rapid appearance of symptoms in calculous ischuria; their tardiness in hysterical ischuria. Innocuousness of symptoms in direct ratio with the quantity of urine secreted. Resistance to inanition in hysteria.

Mechanism of hysterical ischuria. Imperfect supply of information in relation to this subject.

GENTLEMEN: It is my intention to resume and to complete, in our conferences of the present year, the series of studies which we undertook two years ago, and which were rudely interrupted by the painful events with which you are acquainted.

At the epoch when we were forced to separate, I was endeavouring, by the application of preliminary researches concerning *trophic disorders* dependent on nervous influence, to show, as you may remember, that many affections of the muscular system hitherto attributed to peripheral causes, are in reality subordinate to lesions occupying well-defined regions of the gray matter of the spinal cord.

This group of muscular affections, which I propose to call *spinal* *myopathies*, or myopathies of spinal origin, shall occupy our attention in a very special manner. I will also revert to the interesting group of sclerous affections of the spinal cord, and, amongst others, to that sclerosis which determines the symptomatic phenomena of progressive locomotor ataxia.[1] The subject is far from being exhausted, and I shall have occasion to point out, in reference to these diseases, many facts which are new, or which were previously but imperfectly known, and which the investigations conducted in this hospital have made clear.

I intend also to treat of the several kinds of *paraplegia*,[2] produced by slow compression of the cord, of *chronic spinal meningitis*, and of some diseases of the brain and spinal cord, the study of which has been hitherto greatly neglected.

But before returning with you, gentlemen, to these arduous questions, I cannot resist the desire of taking immediate advantage of a number of very remarkable cases of hysteria which are, at present, assembled in our wards. It is requisite to lay hold, without delay, on this fortunate circumstance, for, on account of the mobile character proper to the great neurosis I have named, the symptoms which are, to-day, manifest in a high development may, on the morrow, have completely vanished.

Among these cases there is one especially deserving of attention, which shall form the object of our first conference; it is, if I do not deceive myself, a legitimate example of a rare, an extremely rare affection, the very existence of which is disputed by most physicians.

The study of exceptional cases, gentlemen, is not to be disdained. They are not always mere baits for vain curiosity. Many a time, indeed, they supply the solution of difficult problems. In that respect they may be compared to those lost or erratic species which the naturalist anxiously seeks for, because they show the mode of transition between different zoological families, or enable him to unravel some knotty point of comparative anatomy or physiology.

Hysterical ischuria is the affection to which I allude. At the outset, I should explain to you the technical meaning of this designation, which some of you now, probably, hear mentioned for the first time.

A. Ischuria, stoppage of urine—these terms, technically considered, signify the same thing. Hysterical ischuria has, however, a more restricted meaning.

There is no question here of a simple retention of urine in the bladder—that is a hackneyed fact in hysteria. You know that very commonly the use of the catheter, in such cases, may need to be continued for months, nay years; but then the urine withdrawn from the bladder is abundant in quantity, or, at all events, the amount is not far removed from the normal standard.

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[1] See "Leçons sur les Maladies du Système Nerveux," 2e serie, fascicules 2 et 3.

[2] Charcot, *loc. cit.*, 2e serie, fasc. 1.

In the *ischuria of hysterical patients* the obstruction is situated neither in the urethra nor in the bladder. It lies higher up, either in the ureters or in the kidneys themselves, or still more remotely. But that is a point which yet remains to be decided. The principal fact is this, namely, the quantity of urine secreted in the twenty-four hours and withdrawn by the catheter (for hysterical ischuria is almost always complicated by urethral retention), this quantity, I repeat, is remarkably under the normal amount. It is even frequently reduced to zero, and during several days there is, in fact, absolute suppression of urine.

B. It is proper, in this relation, to establish specific categories. *Oliguria*, or even *total suppression of urine*, may be only a *transient* phenomenon in hysterical cases, and one, as Dr. Laycock has rightly remarked, which may frequently occur unnoticed. Thus, we occasionally observe in hysterical patients, especially at the catamenial periods, a complete suppression of urine which does not last more than from twenty-four to thirty-six hours. There may, perhaps, be some feeling of uneasiness experienced, and the pulse may be quickened; but, after a short time, a few spoonfuls of urine are expelled and the normal state is restored.[1]

The cases upon which I would fix your attention are very different from those to which I have just referred. They present hysterical ischuria at its maximum of development when it has assumed the character of a *permanent symptom*. During the lapse of several successive days, of weeks, and of months, the quantity of urine rendered, in the twenty-four hours, may be quite insignificant in amount or almost nil. Occasionally even, there is *complete suppression* of urine during a series of several days.

When matters take this turn there is superadded, as it were of necessity, another phenomenon which may be called the complement of the first; I mean repeated *vomitings*, which take place daily and even several times a day, so long as the ischuria continues. The ejected matter occasionally, it is said, presents the appearance and exhales the odour of urine. It is certain that chemical analysis has, in two or three cases, detected in this vomited matter *the presence of a certain quantity of urea.*

To sum up, gentlemen, hysterical ischuria offers, in the human species, a more or less exact reproduction of some of the symptoms observed in animals, in cases of nephrotomy or of obliteration of the ureters by ligature.

The experiments of Prévost and Dumas, and particularly those of MM. Claude Bernard and Barreswill, teach us, as you are aware, that, after these mutilations, a vicarious elimination is effected by means of the intestine. In the matter so eliminated, there have been found, according to some observers, *carbonate of ammonia* resulting from the decomposition of the urea (Claude Bernard), and, according to others, *urea* itself has been detected (Munck). However this may be, so long as the elimination is effected, the animals seem to suffer little inconvenience, and it is only when they become enfeebled and the supplementary excretion ceases, that grave phenomena present themselves which soon occasion death.

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[1] Laycock, 'A Treatise on the Nervous Diseases of Women,' London, 1840, p. 229.

You perceive the analogies and, at the same glance, are struck by the contrast. Cerebral symptoms inevitably occur, at a given moment, in cases of experiments practised on animals, whilst in hysterical patients the alternation between renal excretion and vicarious excretion may continue for weeks and months, without any visible disturbance of the general health ever resulting. But I do not wish at present to delay upon this point, to which I will hereafter return.

II.

Such, gentlemen, is hysterical ischuria, at least in its *essential* character, according to the few authors who have acknowledged its existence; for, I repeat, the reality of this disorder has been disputed. You will not find it mentioned in any of the recent works or articles on hysteria, not even in the most complete and most justly esteemed amongst them. There is no mention whatever of it, for instance, in the great work of M. Briquet. In short, of contemporary authors, Dr. T. Laycock, Professor in the University of Edinburgh, is, perhaps, the only pathologist who, in his writings, has given domicile to hysterical ischuria. After devoting to this question a series of articles,[1] in which he relates two original cases, Dr. Laycock returns to the subject in his well known work on the 'Nervous Diseases of Women' (1840). Everywhere else, if hysterical ischuria be mentioned, it receives but a passing notice by way of reference, and not without an ironical allusion to those observers who have been so simple as to gravely accept this "pretended symptom."

On the other hand, it is not uninteresting to note that the physiologists, Haller first, then Carpenter, and Claude Bernard (these, however, without affirming anything), have shown themselves, in reference to this subject, far less sceptical than were such physicians as Prout and R. Willis.

Until recently, I shared the almost general incredulity which prevailed in reference to hysterical ischuria, being, indeed, prepossessed by the teachings of my master, Rayer, who never lost an opportunity of expatiating lengthily on the various deceptions of which hysterical patients are guilty. And he did not hesitate to confess that he, who was himself a sagacious and very keen observer, had often nearly fallen a victim to their strategy. Latterly, my opinions have been somewhat modified in presence of the case to which I shall shortly invite your attention.

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[1] 'The Edinburgh Medical Journal,' 1838.

Before placing you in a position to judge for yourselves whether or not my conversion has been too precipitate, let us investigate the principal reasons why certain authors pass over hysterical ischuria in silence, whilst others only mention its name in order to relegate it amongst the number of chimæras.

1st. In the first place, it must be remarked that hysterical ischuria is a rare phenomenon, at least in its very marked form, for it is possible, as we have already said, that slight cases may often be overlooked.

a. Thus, Dr. Laycock, who searched everywhere, could not collect more than twenty-seven cases, only two of which came under his own observation.

b. Let us add that a somewhat rigorous criticism would, most certainly, reduce this number still further. Most of the cases are old ones, dating as they do from the sixteenth and seventeenth centuries, and they do not offer those notes of exactness which we require at the present period. Others are manifest impostures. Who could be got to believe, for instance, that a woman could, in twenty four hours, render through the ear half a gallon of a fluid which, on being analyzed, was found to contain urea? And that is not all. This very woman is stated to have, at the same time, ejected a similar fluid by the navel—"spirted out" is the term employed by the author of the report. Yet all these details, and many others besides, are recorded, with the utmost gravity, in the 'American Journal of Medical Sciences' (1828). Permit me, I beg of you, to pass in silence over the name of the physician who made himself responsible for this case.

2d. This leads me to say a word on *simulation.* You will meet with it at every step in the history of hysteria, and one finds himself sometimes admiring the amazing craft, sagacity, and perseverance which women, under the influence of this great neurosis, will put in play for the purposes of deception—especially when a physician is to be the victim. As to the case in point, however, it does not seem to me demonstrated that the *erratic paruria* of hysteria has ever been wholly simulated and, as it were, created by these patients. On the other hand, it is incontestable that, in a multitude of cases, they have taken pleasure in distorting, by exaggerations, the principal circumstances of their disorder, in order to make them appear extraordinary and wonderful.

The following is the general sequence of things. Anuria or ischuria, with vomiting, exists alone for a certain time, and the phenomenon is consequently reduced to its simplest form. Soon, however (especially if the symptoms seem to excite the interest and curiosity of the physicians), pure urine will be thrown up in considerable quantity; it will issue from the ears, the navel, the eyes, and even from the nose, as we read in the account given by the American journal. Finally, if the wonder of the observers be extremely excited, there will probably be vomiting of fecal matters.

Amongst this class of cases, that which in France attained most notoriety was the case of a patient named Josephine Roulier, who, for over fifteen months, was a prominent personage in the clinical wards of Professor Leroux. This happened about the year 1810. The patient had first presented the symptoms of simple ischuria with erratic paruria. Nysten, who records the case, analyzed the vomited matters and detected the presence of urea. Shortly after, there came a flow of urine from the navel, the ears, the eyes, the nipples, and finally an evacuation of fecal matters from the mouth. You see, gentlemen, that the sequence is always the same, whatever be the country or century in which the observations were taken. The fraud was discovered by Boyer. It was found that the use of a strait waistcoat caused cessation of the extraordinary phenomena, and some hard fecal pellets were discovered, stored away in the patient's bed ready for use. Unfortunately, Nysten had just published his 'Recherches de Physiologie et de Chimie Pathologiques.' It was necessary to make honourable reparation. A note was accordingly inserted in the 'Journal Général de Médecine,' and another was appended to some copies of Nysten's book.

In presence of these facts, must we conclude that all is imposture in hysterical ischuria? I do not believe it, gentlemen, and I hope you will share my opinion when you shall have been made acquainted with all the peculiarities of my patient's history.

There is another circumstance which is, also, well adapted to throw an unfavourable shadow upon accounts of cases of hysterical ischuria. It is this: apart from hysteria, suppression of urine if it but persist beyond a few days, say three, or four, or five, is an exceedingly serious symptom, which almost necessarily terminates in death.

Leaving aside those cases of anuria depending on an acute or chronic form of Bright's disease, which are too complex for admission here, I will select as typical the *calculous obliteration of the ureters,* supervening in persons previously in good health. In such cases, it has happened that sometimes one kidney has been reduced, through antecedent disease, to a fibrous husk filled with cysts, and consequently rendered incapable of fulfilling its function of eliminating urine; sometimes, but more rarely, both ureters are obliterated at once. It little matters, in so far as our object is concerned, whether this obliteration be accompanied or not by the pains of nephritic colic. Now, Halford,[1] Abercrombie, and all authors who have studied these cases agree in stating that if anuria persist beyond four or five days, comatose symptoms, with or without convulsions, inevitably appear and are soon followed by death.

Life is prolonged a little if even a small quantity of urine can be rendered, but the final result is always the same.

There is, however, the chapter of exceptions, which we should the less neglect, because we are taking advantage of some of its contents. The following exceptional cases are in the books:—

1st. In the case reported by Dr. Laing, of Fochabers, quoted by Robert Willis,[2] anuria lasted ten days, and recovery took place.

2d. In the case of a patient of Dr. W. Roberts (of Manchester) somnolence did not supervene until the eighth day, four days before death.[3]

3d. The most remarkable example within my knowledge of prolongation of life, under such circumstances, is that which was recently published by Mr. Paget, in the 'Transactions of the Clinical Society of London.'[4] Although there was absolute anuria, the comatose symptoms did not present themselves until the fourteenth day. On the fifteenth the patient passed a certain quantity of urine. There was, however, aggravation of symptoms, and the fatal termination took place on the twenty-third day.

However it be, you observe that, just as happened in the case of experiments made on animals, here also the contrast is striking between *calculous ischuria*, which almost certainly kills the patient, and *hysterical ischuria*, which allows the patient to survive during long months without seriously disturbing the general health. We are thus placed in presence of a grave problem. Is it really insoluble? This is a question which we purpose investigating at a future opportunity.

III.

It is full time, gentlemen, that we should enter upon a study of the clinical case which constitutes the basis of our conference. In the first place we must examine the groundwork to which our observations relate; and, with this object in view, the best thing I can do is to show you the patient, and to point out before you the symptoms that are actually in existence, amongst which you will discern the signs of an intense, inveterate hysteria, marked by a characteristic reunion of *permanent symptoms.*

Justine Etch—, born in the department of the Basses-Pyrenees, is forty years of age. She followed the profession of hospital nurse. She was admitted into this hospital of La Salpêtrière in 1869, so that we can trace the progress of her malady during four years.

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[1] 'Medical Transactions,' published by the College of Physicians, t. vi, 1820.

[2] "Urinary Diseases," London, 1838, p. 35.

[3] See account of this patient in Bourneville, "Études Clin. et Therm.," &c., p. 175; and Roberts in 'Mouvement Médical,' 1871.

[4] J. Paget. "Case of Suppression of Urine very slowly fatal." In 'Transactions of Clinical Society of London,' t. ii, 1869.

What is her present condition? That which first strikes you in reference to her is the vast *contracture* which affects the upper and lower limbs on the left side. This contracture, which ceases neither during natural sleep, nor during sleep induced by chloroform unless the influence of this agent be pushed to an extreme, suddenly developed itself on the 20th March, 1870, after a severe hysterical fit. We should mention, however, that the arm had previously been quite paralyzed, but flaccid, whilst the corresponding lower extremity was already rigid. The latter circumstance, together with the rapidity with which the contracture made its appearance, authorized us in declaring, at the time, that no circumscribed cerebral lesion had occurred to cause the phenomena displayed.

Another distinguishing feature which we find, in this patient, is the existence of complete *hemianæsthesia*, which occupies both the contractured limbs and the left half of the face and body. Not only does this anæsthesia affect the external tegument, but it also extends to those portions of the mucous membrane and of the organs of sense situated on the left half of the body. Thus, with respect to the faculty of sight, we note here the existence of *hemiopia* and of *achromatopsia;* this phenomenon was observed by Dr. Galezowski under similar circumstances, and is one to which we shall again return.

When developed to this extent, hemianæsthesia gives us a group of symptoms which are almost specific; I say *almost*, but not *absolutely*, specific, because we shall soon see that even coarse cerebral lesions, confined to certain portions of the encephalon, may at least partially reproduce them.

One other very important symptom is presented by Justine Etch—. This is a pain seated just above the left groin. M. Briquet has given this the name of *cœlialgia*, and regards it as residing in the muscles. For my part agreeing on this point with Négrier, Schutzenberger, and Piorry, I believe its source to be the *ovary*. Whatever be its exact seat, this pain, which I shall term *ovarian hyperæsthesia*, is to a certain extent pathognomonic. Pressure, which exasperates it, causes irradiated sensations of a perfectly special character. These sensations, springing from the ovarian region, successively attain: 1st. the epigastrium; 2d. the neck or throat,—manifesting themselves in these regions by a more or less considerable oppression, the well-known sensation of a ball or globe (*globus hystericus*); 3d. the head where the irradiation is characterized by buzzing and whistling in the left ear, by cephalalgia with throbbings, which the patient compares to so many hammer-strokes on the left temple, and finally to an obnubilation of sight in the corresponding eye. I confine myself, for the moment, to a simple enumeration of these phenomena, which require a more minute description.

Among the other symptoms, I must not forget to mention *retention of urine* and *tympanites*, which also are permanent phenomena in this case. Finally, this woman is subject to special seizures, which are sometimes tetaniform, sometimes epileptiform, but which occasionally resemble the common hysterical type. Thus, this morning, you can perceive a symptom which dates from a seizure that took place two days ago. I refer to the *trismus* which has proved an obstacle to natural alimentation since that period.

IV.

The patient may now retire. We shall be able to relate more fully, in her absence, the other peculiarities of her history. It is a real Odyssey. Hence I shall be often compelled to abridge it, whilst taking care, however, to indicate the sequence of the accidents.

The first fit of convulsions occurred in 1855. What the circumstances were we do not know. The account she gives is quite a romance, a case of rape (?),—a tangled story, the accuracy of which it is difficult to ascertain. It is, however, certain that the seizure in question was apparently one of extreme violence; the patient fell into the fire, burning her face, and you have noticed the indelible stigmata which were caused by that accident. Dating from that period, the fits have continued to show themselves, from time to time, with the same characters, but rather rarely,—about twice or thrice a year.

Ten years later, retention of urine appears. The patient has an attack of hemiplegia with flaccidity of the left side, after one of her usual fits, and is brought into M. Lasègue's wards.

Admitted the same year (1869) to La Salpêtrière, we note—1st. The existence of left hemiplegia, with flaccidity of the upper and contracture of the lower extremity. 2d. Hemianæsthesia and achromatopsia of the same side. The symptoms then presented by Justine Etch— are detailed in the theses of MM. Hélot and Berger.

In 1870 affairs remain much in the same state, only that a new fit has been followed by contracture of the left arm; and, at that time (in 1870) I showed you this patient as supplying an example of the hemiplegic form of hysterical contracture.[1]

In the month of March, 1871, a fit is followed by flaccid hemiplegia of the *right side*. In a month's time, the flaccidity is replaced by contracture. In April, then, we had under our eyes as intense a contracture of the four extremities as it is possible to conceive; the contracture was absolute, persisting night and day, during sleeping and waking, and even resisting the influence of sleep induced by chloroform, or only giving way before the highest dose.

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[1] The lecture referred to, which will be found further on, was first published in the 'Revue Photographique des Hôpitaux de Paris,' 1871, p. 103. Plate xxv of the 'Revue' gives a portrait of this patient.

This woman was, therefore, you perceive, condemned to complete confinement in bed; it was impossible for her to use her limbs. Better conditions could not be desired to render surveillance easy. I took care, moreover, to place near her two devoted patients, bedridden like herself, who were ready to reveal all if they should discover any trickery. I had there the best possible police, that of women over women, for you are aware that if women enter into any plot among themselves they very seldom succeed. This statement will, I believe, be sufficient to convince you, gentlemen, that, during this first period, simulation was impossible. My friends Professors Brown-Séquard and Rouget, who saw the patient at this epoch, declared themselves satisfied with all the precautions taken.

It now remains for us to show you how, in the midst of conditions so favourable to regular inspection, the phenomenon of ischuria was produced.

The ischuria commenced in the month of April, 1871. Before this a woman employed in the hospital, who catheterized the patient, several times a day, had already noticed that the quantity of urine withdrawn was occasionally very minute, whilst at other times none was yielded for two or three days, or even more, nor were the sheets ever moistened.

In addition to these symptoms, which persisted during May and June, vomiting soon appeared, and proceeded without effort or straining. I pretended, from the outset, to be nowise surprised at these occurrences, confining myself to directing that the patient should be discreetly watched both night and day. She was never, however, for a moment detected in any deception.

Cast your eyes, I pray you, on these diagrams which I place before you and you can follow, in the several phases of their evolution, the accidents which have presented themselves to our observation. The tabulation begins on the 16th July, 1871, when I caused the urines and vomited matter to be collected separately, day by day. It ceased in October of the same year.

From the 16th to the 31st of July the quantity of vomited matter varied from 500 to 1750 centilitres, the daily average being one litre (or 1¾ pints nearly). The quantity of urine varied between 0 and 5 grammes; the average in the twenty-four hours being 2.50 grammes (or about 38½ grains). During this period, there was absolute ischuria every second day.

In August, the average amount of urine rendered was 3 grammes (or 46⅓ grains); that of vomited matter was one litre in the twenty-four hours. During the course of this month there was on several occasions, complete anuria, lasting for several days. But, note that the total absence of urine never persisted beyond eleven days.

From the 1st to the 30th September, the average amount of vomited matter rose to a litre and a half (say 2⅔ pints) per day, whilst that of the urine remained at 2.50 grammes.

There is one fact brought into prominence, on examination and comparison of the curves recorded on this table, namely, that the curve of the vomiting generally rises when that of the urine falls, and inversely. Owing to this state of alternate equilibrium there has been a tolerably fair balance maintained between the results of these two phenomena.

What has been the general condition of the patient, as regards health, during this long period of four months which the investigation lasted? At no time have we remarked any disturbance of the general health worth noticing. The alimentation was, as you can readily understand, very limited; the stomach rejected almost immediately, but without fatigue (a characteristic of hysterical vomiting justly noted by Dr. H. Salter[1]), the greater portion of the food swallowed by the patient. Yet, notwithstanding these adverse conditions, nutrition scarcely suffered. This, indeed, is a fact well known, apart from anuria, in cases of uncontrollable hysterical vomiting.

I had believed from the outset that the matter vomited by our patient must contain urea. The first investigations undertaken with a view to detect its presence were, however, fruitless; then I requested the assistance of M. Gréhant, whose competency in such matters is beyond a doubt. He most obligingly placed himself at our disposal.

Twenty-two cubic centimètres of urine collected on the 20th of October, and representing the whole amount of urine rendered that day, gave, on analysis, 0.179 gramme (nearly 2¾ grains). On the 11th October the total vomited matter amounting to 1460 cubic centimetres, gave 3699 grammes (or 26⅕ grains) of urea.

In order to determine if our patient's blood contained a greater proportion of urea than in the normal state, we decided to abstract a little of the venous fluid. In order to accomplish this operation it was necessary to induce sleep, on account of the obstacles presented by the contraction of the members. The operation having been accomplished, M. Gréhant found that in the blood taken from Etch— the urea amounted to 0.036 gramme per hundred grammes of blood; whilst in that taken for comparative examination from a healthy individual there existed 0.034 gramme per cent. You see that the results of the two analyses are (almost) identical.

Unfortunately for the continuance of our investigations, the administration of chloroform had the effect of greatly modifying the symptoms which we had been watching with so much interest; incontinence of urine followed, lasting for several days. The vomiting, moreover, was suspended soon after, and the urine gradually returned to the normal standard.

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[1] The 'Lancet,' Nos. 1 and 2, vol. ii, 1868.

V.

Such, gentlemen, are the results of the first series of studies which decided us to undertake the restoration of hysterical ischuria to a place amongst clinical realities. The same phenomena, indeed, were destined again to present themselves, under a less striking aspect it may be, but one quite as full of interest. In this second phase, no complete anuria occurred, not even temporarily. We noted a simple oliguria. The matter vomited was not thrown off in such abundance. In a word, if the accidents had been a little less marked, and if we had not been enlightened by the foregoing observation, it is incontestable that the supplemental elimination of urea might have altogether escaped attention.

Let us briefly observe what took place during this second period. After a more or less complete remission of symptoms, retention of urine was the first to make its appearance again. This was in January. The following month, after a fit, we noted alternations of oliguria and of polyuria, when there were two litres (or 3½ pints) passed *per diem*. In March, the urinary secretion diminished to a decided extent; and, on the 18th of the same month, vomiting again showed itself. From that date, until the 31st of March, the daily average of vomited matter amounted to 500 grammes. In April, the average was 800 grammes for the vomited matter and 100 grammes (=1543.23 grains) for the urine.

During this new phase, in which the disease offered itself for investigation, we were not under so favourable conditions as on the previous occasion. The patient's right arm had become nearly quite free. Hence it was urgent that we should protect ourselves against every possible cause of error.

In addition to the customary surveillance, which was not relaxed for a moment, we had recourse to the following precautions: the patient's bed was carefully examined from time to time, and neither vessels nor catheters were allowed to remain at her disposal. Finally, I succeeded in persuading her that it might, perhaps, be beneficial on account of the contracture that still persisted in the left arm to make use of a strait jacket. To this she consented. The application of the strait jacket was not, however, absolutely continuous; it was removed at meal times, when the patient was watched by the person who fed her.

At different periods, during the month, M. Gréhant analyzed the urine and the vomited matter rendered in twelve days. During this lapse of time, the daily average of the urine was 206 grammes, cantaining 5.09 grammes of urea. The daily average of vomited matter, amounting to 362 grammes, yielded 2.138 grammes of urea. Adding together the two totals of urea rendered, we obtained the low figure of 5.233 grammes. I can show you a specimen of the oxalate of urea which M. Gréhant extracted from the matter vomited during the four and twenty hours. We shall make use of this result in a moment.

We did not discover, any more than on the previous occasion, the existence of a supplemental elimination by the skin or intestines. The patient is habitually constipated, and we did not observe, at either time, any peculiarity in relation to the external tegument. Her general health has not undergone any noteworthy alteration, and the temperature never was above 37° C. (=98.6° F.) and some tenths.[1]

Thus, gentlemen, this new investigation confirms the accuracy of the first, and both concur to establish the existence of *hysterical ischuria*, with *erratic paruria* as a proven pathological phenomenon, utterly beyond the possibility of simulation. If this conclusion be legitimate, it is obvious that some value must be attached to the records of former cases. It is only requisite to separate the truth from what is fictitious, to eliminate, for instance, certain extraordinary phenomena such as the flow of urine from the nose, eyes, &c., and the vomiting of fecal matter. Some of these accounts, indeed, exhibit, in all their details, the characteristics of a truthful statement. We would place in this category the case reported by Dr. Girdlestone (of Yarmouth), and some others besides.

VI.

I desire now, gentlemen, to investigate with you, whether the contradiction we noticed between *ordinary anuria* observed in man, or *experimental anuria* produced in animals, on the one hand, and *hysterical ischuria*, on the other, be quite as absolute as it seemed at first sight.

In the first group of cases, death is nearly certain to happen within a brief space; in the second, the general health is kept up in a tolerably perfect state for an indefinite period. The opposition is therefore extremely marked. Is it not, however, possible by a careful scrutiny of all the circumstances, to arrive at the reason of this discordance? I am far from being in a position to solve the problem in a decided manner. Hence, I must content myself with stating an hypothesis which, perhaps, may seem plausible to you, but which I request you, in any case, to take only for what it is worth.

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[1] Etch— has presented, during the present year (1875), a new period of hysterical ischuria. On examining the table, which represents the quantity of urine rendered each day, and the results of 112 chemical analyses made by M. P. Regnard, we notice that, during three months, the patient rendered from fifteen to twenty grammes of urine daily, containing from three to four decigrammes of urea. Some days, however, in the midst of painful fits, the patient in a few hours emitted up to four litres of urine, containing 27 grammes of urea. During this period, Etch— had no vomiting, by which urea might have been evacuated, as happened on the occasions specified in the lecture. (See in reference to this communication which we made in conjunction with M. P. Regnard to the 'Société de Biologie,' 3 Juilliet, 1875). We shall have occasion, further on, to mention under what circumstances this ischuria suddenly ceased. (See Lecture XII.)

That animals should always succumb after nephrotomy, or permanent ligature of the ureters, is very natural. But we have a right to ask what would happen if an experiment could be made in which, for instance, the obstruction of the ureters, instead of being permanent, was intermittent? Would existence be prolonged if, in such conditions, a regular alternation were established between the renal and the supplemental elimination? In spite of the interest which would attach to the solution of this problem, I put it aside in order to come to the question of human pathology.

Let us then resume the consideration of calculous obstruction of the ureters, to which we referred above.

The first remark which suggests itself to the mind is the following: In our patient, complete anuresis never lasted longer than a period of ten days. Now, according to what we have mentioned, that is not the extreme limit at which, in obstruction of the ureters, the symptoms of uræmic poisoning necessarily appear, since, in Paget's case, the patient preserved his general health and functional integrity till the fourteenth day. No doubt the quantity of urine secreted by Etch— on alternate days was very minute, but small as it was, it is of real importance; for every author, since Halford, has recognized the great relief and improvement which ensues in the ureter-ischura of calculous patients, on the emission of the smallest quantity of urine.

Again, we have another peculiarity:—calculous patients are suddenly seized, surprised as it were, in the very midst of good health, whilst, to judge from my own observations, hysterical ischuria only arrives gradually at its apogee. Perhaps, there is a question of *habit* here, of which it is well to take heed. Far be it from me, however, to suppose that hysterical patients enjoy a peculiar immunity, a kind of Mithridatism, with respect to uræmic intoxication. The resistance they present, in the conditions we are considering, is most probably due to another cause—the question here is rather one of doses. Let me explain.

The insignificant quantity of urea eliminated by our patient, both in the urine and the vomited matter, must have caught your attention. During a period of twelve days, we have said, she only got rid of five grammes of urea daily. This amount is you are aware, far below what Schérer found in the case of an insane man, who had been fasting for three weeks—from nine to ten grammes of urea was the quantity found in that instance.[1] We have also seen that there was no reason for believing that, in our patient, there was any elimination of urea by the perspiration[2] or the stools. Now, in every case of poisoning, and uræmia is probably no exception to the rule, we must take the *dose* as an element to be considered.

Is it not, then, most likely that this very diminution of the amount of urea—which doubtless corresponded to a correlative diminution of extractive matters—should account, in our patient, for the absence of every symptom of uræmic intoxication? We are thus induced to admit that in this case of Etch— there was, so long as the ischuria lasted, a decrease of activity in the phenomena of disassimilation, manifesting itself by an absolute diminution of excrementitious matter.

This condition, besides, is probably common to a group of hysterical cases. In fact, it has been long remarked that some of these patients when subject to *uncontrollable vomiting*, bear up wonderfully against the influence of a restricted and insufficient alimentation, without losing their plumpness or suffering any disturbance in their health worth noticing.[3] It would certainly be of interest, under such circumstances, to make comparative analyses day by day, of the blood and urine, in order to ascertain the amounts of urea and extractive matters present. We might, possibly, by this means obtain the solution of the problem which I can only indicate here.

VII.

What is the mechanism on which hysterical ischuria depends? What is the seat of the obstacle which hinders the secretion of urine from being effected? The urethra and the bladder are evidently not incriminated. Is the obstacle in the ureter, or in the kidney itself? No reason exists for suspecting the existence of phlegmasia of the renal glands or the ureters; the composition of the urine, and the other symptoms, likewise, are opposed to such an hypothesis. It is more allowable to consider that we have here to deal with some action of the nervous system. The influence of the nervous system on the secretion of urine is not a doubtful matter. It may suffice to remind you, by way of illustration, that temporary suppression of urine may be produced in dogs by the mere fact of laying open the abdomen, as M. Claude Bernard has observed; and that, in the operation for vesico-vaginal fistula, it also occasionally happens (as Jobert de Lamballe remarked) that the urine may be suppressed for a certain period.

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[1] It is proper to mention that the difference of sex and condition may have contributed somewhat to this disparity of results. With respect to sex, Beigel taking an average of 58 analyses, found that 35.6 grammes of urea were yielded by a male in the twenty-four hours, whilst a female yielded only 27.6, in the same time. It must also be remembered that Etch— was in enforced inaction, during the investigation; and that she seems to have been unable to receive much nutritious food. Lehmann, who found 53.19 grammes of urea ih the urine rendered on the eighth day of a strictly nitrogenous diet, was able to reduce the amount to 15.41 grammes, by living for eight days on non-azotized food. These facts go to support Professor Charcot's views. (S.)

[2] According to the researches of M. Favre there are in the normal state only 0.43 gr. of urea in 10,000 grammes of perspiration. (S.)

[3] A singular case of this kind is mentioned by Sir T. Watson ('Lectures on the Principles and Practice of Physic,' vol. i, art. 1, p. 704, London, 1857). "A romantic girl," he says, "was for some months under my care in the hospital with that complaint (hysterical hæmatemesis). She vomited such quantities of dark blood (which did not coagulate, however), as I would not have believed if I had not seen them. Day after day there were potfuls of this stuff, yet she did not lose flesh, and she menstruated regularly; and what was very curious, the vomiting was always suspended during the menstrual period and recurred again so soon as the natural discharge ceased. . . . At last I sent her away just as bad as when she entered the hospital." She recovered afterwards on getting married. The alternation here may be compared with that pointed out by M. Charcot. (S.)

May it not be that, in the case of our patient, there existed a spasmodic obliteration of the ureters? That these conduits possess very marked contractile properties is an admitted fact: thus, Mulder observed them contracting energetically in a patient suffering from ecstrophy of the bladder, and Valentin reports having noticed a very decided contraction supervening under the influence of irritation of the nervous centres.[1] This supposition appears to be supported by analogical reasoning, for in hysterical patients we frequently find long-continued contraction of the tongue, the œsophagus, &c. Hysterical ischuria should, consequently, be paralleled with calculous obliteration of the ureters. Unfortunately, there are objections of some weight to be alleged against this view.

The experimental researches of Herr Max Hermann demonstrate, you are aware, that the proportion of urea to the quantity of urine voided, diminishes when there is counter-pressure of the ureter. If the pressure reach 0.060 millimètres of mercury no more urea is found.

Mr. Roberts (of Manchester)[2] confirmed the accuracy of this

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[1] Donders' 'Physiologie.'

[2] "The Pathology of Suppression of Urine," in 'The Lancet,' 1868, May 23 and 30,—1870, June 18; 'Mouvement Médicale,' 1871, pp. 22, 32, 128.

Since this Lecture was delivered by M. Charcot, M. Ch. Fernet has communicated to the Société Médicale des Hôpitaux a note entitled "De l'oligurie et de l'anurie hystériques et des vomissements qui les accompagnent" ('Union Médicale,' 17 Avril, 1873, p. 566). After having stated M. Charcot's opinions, M. Ch. Fernet reports an interesting case, of which the following is a summary:—

Marie L—, aged 19 years, chloro-anæmic, menstruated at sixteen years. Her menstruation has always been very irregular. A sister of the patient is subject to frequent hysterical seizures. In January, 1871, Marie L— got a fright, which threw her into an hysterical fit. In May, extreme debility, uneasiness, pains in the limbs (strengthening regimen, bark, iron, sea-bathing). At the end of the month of August, after a sea-bath, Marie L— was taken for the first time with vomiting. "She began by throwing up the solid food, then, after a few days, she vomited all she took. . . . These repeated vomitings continued, without intermission, till the month of October, then subsided for a fortnight, when they recomstatement in its relation to man. In a case of calculous obstruction of the ureter, there escaped a small quantity of clear urine, con-

menced with their original intensity and persisted without respite. . . . In March, 1872, she was admitted to the Hôtel-Dieu (under M. Moissenet's charge).

Treatment.—Cold lotions, ice and champagne, blister with morphia, to the epigastrium. The vomitings gradually diminished, and only appeared again at intervals: the patient left the hospital on the fifteenth of April, the vomitings having ceased. During the months of May and June, rare vomitings. They returned in July, after some vexations, and again stopped, owing probably to the influence of bromide of potassium. At the end of July, another emotional disturbance set them again in action with their former frequency and persistence.

Marie L— was admitted a second time to the Hôtel-Dieu, on the 18th of August, 1872. She then came under M. Ch. Fernet's observation, when she presented the following symptoms: excessive debility; anæmia very marked, characterized especially by discoloration of the skin and mucous membranes; intercostal neuralgia; ovarian sensibility developed on the left side, painful on pressure; anæsthesia existing on different points of the skin; complete plantar anæsthesia; profound analgesia of the superior extremities; achromatopsia of the left eye, which cannot distinguish yellow tints; vomitings. The patient alleges that, since their appearance, she only voided a very minute quantity of urine, that she often remains for several days without voiding even a drop.

September 4.—Milk-diet, exclusively. From the 4th to the 9th September, there was but one emission of urine (about 150 grammes). From this epoch, M. Ch. Fernet caused to be exactly weighed, 1st the quantity of food taken, and 2d the amount of urine voided and of matters vomited, and, after recording these quantities day by day, he says: "The examination of this table allows us to establish a close connection between the state of the urinary function and the vomitings. In a first period, comprised between the ninth and sixteenth September, that is to say, eight entire days, the urine was completely suppressed during the first six days, and its quantity was very scanty during the last two; now, in this period, the patient, being on a milk-diet, vomited a quantity of liquid matter, at first equivalent to one-half or three-fourths of the fluids swallowed during the first four days, then a quantity perceptibly equal to the quantity of milk taken during the last four days.

"In a second period, comprising nine days (from the 18th to the 26th Sept.), the quantity of vomited matters seems to have diminished; but this is not exactly the case, as we see on comparing it with the quantity of food taken. The diet having been changed, and being now composed of cold soup (*bouillon*), of raw beef, and of lemonade, the vomited matter still represents almost the whole of the ingesta. Now, during this time, there was a little urine the first two days (15 grammes and 250 gr.), but its emission was suspended during the seven days following.

"Finally, in a third period, lasting four days (from the 27th to 30th Sept.), we see the urinary function re-established, and the amount of urine reaching the normal standard (1000 grammes, 500 gr., 1100 gr. the last two days): at the same time, the vomitings diminished on the second day and subsided on the third and fourth."

Desirous of ascertaining whether, as indicated by M. Charcot, the vomiting might not be attributable to the supplemental elimination of urea by the stomach, M. Ch. Fernet requested M. E. Hardy to analyze the urine and the vomited matter. From a summarized table of these analyses, it appears that "the urea was always present to a noteworthy amount (from 0.55 gr., to 1.87 grammes) in the vomited matter; also, when the secretion of urine was suppressed, the quantity of urea contained in the vomited matter was gradually increased during that space of time. From the 17th to the 27th Sept., the quantity rose from 0.62 to 1.08 grammes. Finally, from the day when the urine issuing from the bladder reached what might be considered a normal amount, the urea diminished in the gastric secretion, disappearing doubtless at the same time as the vomiting."

A moral influence—the administration of the pills termed "*fulminantes*" (*mica* taining only 50 centigrammes of urea per 1000 grammes. Now, in the case of our patient, the urine contained 15 grammes of urea per 1000 grammes,—an amount approximating to the normal standard.

Judging from this, gentlemen, the obstacle in hysterical ischuria would not lie in the ureters. Where then does it reside? Should we invoke an influence of the nervous system analogous to that which Ludwig discovered in the case of the salivary gland? In the absence of all information on this point, we are compelled to leave the question in suspense.

LECTURE X.

HYSTERICAL HEMIANÆSTHESIA.

SUMMARY.—Hemianæsthesia and ovarian hyperæsthesia in hysteria. Frequent association of these two symptoms. Frequency of hemianæsthesia in hysterical patients; its varieties, complete or incomplete. Characters of hysterical hemianæsthesia. Ischæmia and the "Convulsionnaires." Lesions of special senses. Achromatopsia. Relations between hemianæsthesia, ovarian hyperæsthesia, paresis and contracture. Variation of symptoms in hysteria Diagnostic value of hysterical hemianæsthesia; necessary restrictions.

Hemianæsthesia depending on certain encephalic lesions. Its analogies with hysterical hemianæsthesia. Cases in which encephalic hemianæsthesia resembles hysterical hemianæsthesia. Seat of the encephalic lesions capable of producing hemianæsthesia. Functions of the optic thalamus; British theory; French theory. Criticism. German nomenclature of different parts of the encephalon. Its advantages as regards the circumscription of lesions. Cases of hemianæsthesia recorded by Türck; special seat of the encephalic lesions in these cases. Observation of M. Magnan. Alteration of special senses.

GENTLEMEN,—There are two points in the history of hysteria, upon which I wish to lay particular emphasis, in this and the following lectures. These are, on the one hand, *hysterical hemianæsthesia*, and on the other, *ovarian hyperæsthesia*. If I set these two

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panis)—caused a sudden change in the condition of Marie L—, dating from the 17th September. The vomiting ceased, the secretion of urine resumed its course. Finally, the patient left the hospital, in very fair health, in the course of November. M. Ch. Fernet, in concluding his note, points out the numerous analogies between this case and that of M Charcot's patient.

We may mention also a thesis of M. Secouet, 'Des vomissements urémiques chez les femmes hystériques,' (Paris, Avril, 1873), which contains the report of a case that, though imperfect in some respects, should apparently be classed in the category of hysterical ischuria. (B.)

phenomena side by side, it is because they are generally found associated together in the same patients. With reference to ovarian hyperæsthesia, I hope to render evident to you the influence of *pressure on the ovarian region*—an influence formerly acknowledged, but afterwards denied—over the production of the phenomena of the hysterical seizure. I shall show you that this operation determines, either the premonitory symptoms merely of the hysterical fit, or, in a certain number of cases, the complete seizure. You will thus be enabled to verify the accuracy of the assertion formerly made by Professor Schutzenberger, with respect to this phenomenon, in spite of the contradictions offered by certain observers.

I shall likewise show you a method which I have discovered, or rather re-discovered, which, in the case of some patients, enables us to arrest the course of even the most intense hysterical fit,—I refer to the *systematic compression of the ovarian region.* M. Briquet denies that this compression has any real effect. That is an opinion which I cannot share, and this leads me to make a general remark in reference to M. Briquet's book.[1] The work is an excellent one, the result of minute observation and patient industry, but it has perhaps one weak side; all that relates to the ovary and the uterus is treated in a spirit which seems very singular in a physician. It exhibits a kind of prudery, an unaccountable sentimentality. It appears as though, in reference to these questions, the author's mind were always preoccupied by one dominant idea: "In attempting to attribute everything to the ovary and uterus," he says for instance, somewhere, "hysteria is made a disorder of lubricity, a shameful affection, which is calculated to render hysterical patients objects of loathing and pity."

Really, gentlemen, that is not the question. For my own part, I am far from believing that lubricity is always at work in hysteria; I am even convinced of the contrary. Nor am I either a strict partisan of the old doctrine which taught that the source of all hysteria resides in the genital organs; but, with Schutzenberger, I believe it to be absolutely demonstrated that in a special form of hysteria,—which I shall term, if you please, the *ovarian form*,—the ovary does play an important part.[2] Five patients whom I shall present to you in succession are, if I mistake not, manifest examples of this form of hysteria; you can verify the accuracy of the description I am about to give, by personal examination.

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[1] Briquet, 'Traité clinique et thérapeutique de l'hystérie,' Paris, 1859.

[2] Grisolle ('Traité de Pathologie Interne,' 9e édit., t. ii, p. 844) mentions the case of a girl, aged 22, who had neither vagina nor uterus, and yet was subject to most violent fits of hysteria. On autopsy, MM. Chassaignac and Prévost could discover no trace of a uterus, but found, in the ovarian regions, two bodies which were apparently the *ovaries.* The patient had, every month, exhibited all the symptoms of pre-catamenial congestion. (S.)

I.

You are all acquainted with the *hemianæsthesia of hysterical patients.* There would be some ingratitude in not knowing the nature of this symptom, for it has been discovered by purely French investigations. Piorry, Macario, and Gendrin, have each of them, in his turn, described it and dwelt upon its characteristics. Not long after them, Szokalsky made it known in Germany; but nothing remained for him to do save to confirm by observations, which are, however, very meritorious, the facts that had been already declared by our countrymen.

In order to keep within bounds, I shall enter upon a discussion of *complete hemianæsthesia* only, such as we find in intense cases. This will be sufficient for my present purpose. Even in the degree mentioned, it is a frequent symptom, since according to M. Briquet, it obtains in 93 cases out of 400. Considered with respect to position, we find, according to the same author, that in 70 cases the left side is affected, and in 20, the right.

You know what happens under such circumstances. Supposing that the two halves of the body are vertically divided by an antero-posterior plane, one entire side—face, neck, body, &c.—will have lost the sense of feeling; and though this loss of sensibility very often affects the superficial parts merely,—the external tegument,—yet it sometimes also invades the deeper regions, affecting the muscles, bones, and articulations

Hysterical hemianæsthesia shows itself, as you are aware, under two principal aspects; it is complete or incomplete. *Analgesia*, with or without insensibility to heat or cold, or thermo-anæsthesia is one of the commonest varieties of this species. The distinct manner in which the anæsthetic parts are separated from the healthy parts is also an important characteristic of hysterical hemianæsthesia. On the head, face, neck and body the demarcation is often perfect and very closely corresponds with the median line. Another symptom, well deserving of mention, is constituted by the comparative pallor and coldness of the anæsthetic side. These phenomena, conjoined with a more or less permanent ischæmia, have been many times observed. Examples of them have been given by Brown-Séquard and Liégeois.[1] A difficulty in inducing bleeding by pricking the anæsthetic parts with a pin may, in intense cases, be a characteristic of the ischæmia in question.

I noticed this peculiarity on a former occasion. The matter came under my observation in this way: on leeches being applied to a patient affected by hysterical hemianæsthesia, I saw that their bites yielded very little blood on the anæsthetic side, whilst on the healthy side it flowed as usual. Grisolle, who, you are aware, was a very wise and exact observer, had noted the same phenomenon.

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[1] Liégeois, 'Mémoires de la Société de Biologie,' 3e série, t. i, p. 274.

This ischæmia which, indeed, is rather rare when so intense, may furnish an explanation of certain reputedly miraculous occurrences. Thus, it is stated, that, in the epidemic of Saint Medard, the sword-blows given to the "Convulsionnaires" did not cause bleeding. The reality of the occurrence cannot be rejected without examination. If it be true that many of these "Convulsionnaires" were guilty of trickery, we are nevertheless compelled to acknowledge, after an attentive study of the question, that most of the phenomena which they presented, and of which history has given us a naive description,[1] were not entirely simulated, but merely amplified and exaggerated. It has been critically demonstrated that hysteria carried to an extreme, was almost always the active agent in these cases; and in order that a wound, such as that made by a sword, should not, when inflicted on these anæsthetic women, have caused bleeding, it was only necessary, as you may infer from what precedes, that the instrument should not have entered too deeply.

There are other characters also of hysterical hemianæsthesia which are deserving of all our attention, from a clinical as well as from a theoretical point of view. The *mucous membranes* are affected, on one side of the body, in the same manner as the external tegument. The organs of the senses themselves are affected to some extent in the anæsthetic side. *Taste* may have vanished in the corresponding half of the tongue, from tip to base. The sense of *smell* is less acute. *Vision* is weakened in a very remarkable manner, and if amblyopia occupy the left side, we may meet with a most noteworthy phenomenon, to which M. Galezowski has called attention, and which he designates by the name of *achromatopsia*. However, we shall return to this topic.

Hysterical *hemianæsthesia* does not seem to affect the viscera. Thus, to mention the ovary merely, we find hyperæsthesia and not anæsthesia present. That organ may be very painful on pressure, when the abdominal wall is perfectly insensible. Now, gentlemen, there exists a most remarkable relation between the position of the hemianæsthesia and that of the ovarian hyperæsthesia. If the former occupy the left side, the hemianæsthesia occupies the left side, and *vice versâ*. When ovarian hyperæsthesia is double, it is the rule that the anæsthesia shall present itself in a generalized form, and it consequently occupies nearly the whole, or quite the whole of the body.

Not only does such a relationship exist between the seat of the hemianæsthesia and that of the ovarian hyperæsthesia, but a similar relationship exists with regard to the paresis, or to the contracture of the limbs. Thus, when the paresis or the contracture supervenes, it always shows itself on the same side with the hemianæsthesia.

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[1] Carré de Montgeron, 'La Verité des Miraoles opérés à l'intercession de M. de Pâris et autres Appelants,' &c., 1737.

The hemianæsthesia, as described, is, in the clinical history of hysteria, a symptom of the greater importance, inasmuch as it is well-nigh permanent. The only variations which it exhibits, are dependent upon degree, on the intensity of the phenomena which constitute it, and occasionally, we should also mention, on the fluctuation of some of these phenomena.

Achromatopsia belongs to the number: it was distinctly and repeatedly observed in one of our patients, a few weeks ago, from whom it has now completely disappeared.

It is necessary to bear in mind that hemianæsthesia is a symptom which requires to be sought for, as M. Lasègue very judiciously remarks.[1] There are, in fact, many patients who are quite surprised when its existence is revealed to them.

II.

I propose now to investigate to what extent hemianæsthesia, such as we have described it, is a symptom proper to hysteria. In reality, it is very rare for it to be reproduced, with the general grouping of all its characteristics, by any other disease. Its well-established existence is, therefore, a valuable indication, one which will often reveal the real nature of many symptoms, which would otherwise remain doubtful. That is a point on which M. Briquet was right to lay great stress. In order to illustrate the importance of this fact, he relates the case of a woman who, after a violent emotion, fell rapidly into a more or less profound coma, with or without premonitory convulsions (*i. e.*, the comatose form of hysteria), and who was seen, on recovering her senses, to be stricken with more or less complete hemiplegia. Here we have a group of symptoms which it is not very rare to meet with in practice, and, on such an occurrence, it may happen that the physician will feel himself placed in a very embarrassing position. Now, the presence of hemianæsthesia, arrayed in all its characteristics which would most probably be found on such occasion, might then, according to M. Briquet, indicate the true path to the observer. This assertion is perfectly accurate; I have no fault to find with it, except as regards one point.

If it be true that hemianæsthesia is an almost specific symptom, inasmuch as it is not found with the same characteristics in the immense majority of cases of material lesions of the encephalon (hemorrhage, softening, tumors), we cannot admit this to be an absolute characteristic. It is, above all, inaccurate to say that *the hemianæsthesia, developed under the influence of encephalic lesions, always differs from hysterical hemianæsthesia, by the fact that, in the*

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[1] 'Archives Générales de Médecine,' 1864, t. i, p. 385.

former case, the skin of the face does not participate in the insensibility, or that, *when it exists, it never occupies the same side as the insensibility of the members.* This is an inaccuracy which has been reproduced, almost in the same terms, in the otherwise very interesting thesis of M. Lébreton.[1]

I feel some repugnance in again attacking the remarkable work of M. Briquet, but the more estimable the work—and it is justly esteemed,—the more serious become any inaccuracies which may have slipped into it. This reflection will, I hope, justify me in criticizing it.

Gentlemen, there are cases, which, though indeed exceptional, are thoroughly authentic, where certain circumscribed cerebral lesions (*en foyer*), may cause the production of hemianæsthesia with all the signs that characterize it in hysteria—*or very nearly all.* Allow me to discuss this subject, in some detail.

The classic doctrine, at least amongst us,—a doctrine which, besides, appeals to the data of clinical observation, and to those furnished by experiments on animals,—teaches that circumscribed cerebral lesions (*en foyer*), which so profoundly affect the power of motion, especially when they occupy the region of the *optic thalamus* and *corpus striatum*, produce but little effect as regards sensibility. From this point of view, gentlemen, the result is said to be always the same, whether the lesions occupy specially the corpus striatum, the optic thalamus, or the rampart of the amygdalæ (claustrum).

At first glance, when in presence of the suddenly developed lesions which determine an apoplectic fit, and which affect any one of the points just enumerated, the symptom which strikes the observer is a hemiplegia, more marked in the upper than in the lower extremity, and accompanied by flaccidity.

In the face, the paralysis usually affects the buccinator and the orbicularis oris; the tongue also is mostly protruded to the paralyzed side. In addition to motor-paralysis comes paralysis of the vaso-motor nerves, manifested by an elevation of temperature in the paralyzed limb. Occasionally, this vaso-motor paralysis makes its appearance from the outset.

As to sensibility, it is not modified in a perceptible manner, or at least not in a *durable* manner. The special senses present no serious alterations, except some complication supervenes, as where *embolism of the arteria centralis retinæ* occurs (in cases of brain-softening consecutive on the migration of a valvular vegetation), or where *compression*, by contiguity, of the tractus opticus, happens (on occurrence of a somewhat voluminous *hemorrhagic* lesion). Such is a summary of the symptoms which are met with in the immense majority of cases of hemorrhage and softening affecting the points of the encephalon we have mentioned.

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[1] Lébreton, 'Des differentes variétés de la paralysie hystérique,' Thèse de Paris 1868.

Undoubtedly, gentlemen, that is what takes place, in the great majority of cases. But, the chapter of exceptions accompanies the rule. There are cases, and I have myself observed several of this kind, in which sensibility is affected in a predominant manner, and in which anæsthesia persists, even after the recovery of motion.

Such alterations of sensibility may present themselves with the following characters. The anæsthesia affects one entire half of the body and stops just at the median line. The corresponding half of the face, both as regards the skin and the mucous membranes, shows insensibility, exactly as in hysterical hemianæsthesia. Then also *analgesia* and *thermo-anæsthesia* may be observed, with conservation of tactual sensibility, as MM. Landois and Mosler[1] have ascertained. Finally, there are also cases, though of more rare occurrence and as yet imperfectly described, but having still their own importance, which render it probable that, under such circumstances, alterations of the special senses may exist on the side opposite to the encephalic lesion, or, in other words, on the same side with the hemianæsthesia.

The physicians of the last century have already remarked these exceptional phenomena. Borsieri, among others, relates the history of a patient who, three months before, had been stricken with apoplexy, and in whom anæsthesia still persisted, although the power of movement had returned. He quotes some other cases of the same kind from different authors.[2]

Analogous cases have been mentioned by Abercrombie, Andral, and, in later days, by Hirsch,[3] Leubuscher, Broadbent, Hughlings-Jackson,[4] and especially by Türck. The latter alone has been able to furnish decisive data in reference to the position occupied by the encephalic lesions in such cases.

When the hemianæsthesia presents itself with these characters, the optic thalamus is almost always affected in a predominant, if not in an exclusive manner. For my own part, I have seen hemianæsthesia superadded to hemiplegia, in many patients affected with cerebral hemorrhage, and, in such cases, on post-mortem examination I always found the lesion of the optic thalamus, the existence of which during life I had ventured to announce.

From what precedes, gentlemen, should we conclude that the lesion of the optic thalamus is the real organic cause of the hemianæsthesia observed in all these cases? That is a question deserving of discussion. I am thus led to speak of the physiological theory, which may be called the *British theory*, because it was, I believe, first published and maintained by Messrs. Todd and Carpenter, two British authors. According to this theory, the *optic thalamus* is the centre of perception of tactual impressions; it would, in some degree, correspond to the posterior cornua of the gray substance of the spinal cord. The *corpus striatum* would be the terminal of the *motor tractus* and connected with the execution of voluntary movements; it would be analogue of the anterior cornua of the cord.

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[1] Landois et Mosler, 'Berliner Klin. Wochens.,' 1868, p. 401.

[2] Borsieri, 'Inst. pract.,' vol. iii, p. 76.

[3] Hirsch, 'Klinische fragments,' I. Abth., p. 207, Kœnigsberg, 1857.

[4] H. Jackson, 'Note on the Functions of the Optic Thalamus.' In 'London Hospital Reports,' 1866, t. iii, p. 373.

This theory of which Schrœder van der Kolk[1] has shown himself the avowed partisan, is, if we might use the word, the antipodes of the French theory, which you will find set forth in a very complete manner in M. Vulpian's Lectures. According to the latter view, the centre on which sensitive impressions are transformed into sensations would not be in the brain proper, because an animal, from which the brain, including the optic thalamus and the corpus striatum, has been removed, continues to see, to hear, and to feel pain, &c. The centre of sensitive impressions would therefore reside lower down, in the protuberantia and perhaps also in the crura cerebri.

Under this hypothesis, the following is the manner in which its advocates regard, in the pathological domain, those authentic facts which show a lesion of the optic thalamus coinciding with the decrease or abolition of sensibility on the side of the body stricken with hemiplegia. They say, and their allegation is perfectly correct, that, in such cases, we have frequently to do with recent lesions, such as *intra-encephalic hemorrhage*, or *ramollissement*, or *tumours*—lesions by which the optic thalamus is extremely distended and which, consequently, may have the effect of determining the compression of the adjacent parts,—of the crura cerebri for instance. It is, on the other hand, well established that, in a number of cases, the optic thalamus may be injured, even gravely and throughout a large portion of its extent, without being followed by any special disorder in the transmission of sensitive impressions.

To the last argument, the British authors, M. Broadbent[2] among others, oppose the plea that the optic thalamus, the presumed centre of sensitive impressions, should doubtless be assimilated to the gray axis of the spinal cord—the latter, it is known, continues to transmit these impressions even when it has suffered the most serious derangement, if only a small remnant of gray matter persist, capable of connecting its lower with its upper extremities. I confess that the comparison seems to me far-fetched, especially from the moment it is laid down as a principle that the optic thalamus should be considered a centre; for, so far as regards the transmission of sensitive impressions, the gray axis of the cord is manifestly merely a conductor.

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[1] Schrœder van der Kolk, 'Pathol. und Therapie der Geistenkrankheiten.' Braunschweig, 1863, p. 20.

[2] Broadbent, 'Medical Society,' London, 1865, and 'Med.-Chir. Review.'

However this may be, gentlemen, such is the state of the question. In my opinion, the disputed points cannot be definitely solved, except by means of careful clinical observation, verified by studious anatomical investigations, the chief aim of which should be to establish, with great precision, the seat of the encephalic lesions, to which the symptoms recorded during life might be correlated. And the circumstances of the case should be such that the influence of compression, or any other phenomenon, acting by contiguity, would be completely eliminated. Now, gentlemen, in the present state of the science, the cases which include all these conditions are extremely rare, so far, at least, as my knowledge goes. We may, however, mention as approximating to this ideal, the cases which were presented by L. Türck to the Academy of Sciences of Vienna,[1] to which I have already alluded. They were four in number.

In the instances recorded by L. Türck, there had been, gentlemen, either old hemorrhagic foci, then represented by ochreous cicatrices, or ramollissement foci arrived at the stage of cellular infiltration. In all the cases, the hemiplegia resulting from the presence of foci had disappeared long before death, but the hemianæsthesia had persisted until the fatal end. The portions of the encephalon affected by the alteration are carefully mapped out.

The German nomenclature of the different parts of the encephalon, however forbidding it seems to us, on account of its multiplicity of strange terms, yet presents in my opinion, an incontestable advantage, that, namely, of supplying a very complete topographical map, if I may make use of the comparison, where the smallest hamlet receives a name. The French nomenclature has, no doubt, the advantage of tending to simplification, but this is sometimes to the detriment of absolute exactness; it is often incomplete. Now, with respect to the question which occupies us, there is no detail, however minute, which ought to be neglected. We must, at all hazards, take heed of the slightest details, for we are quite ignorant, in the actual state of the science of the brain-physiology, whether some little point, which has no name in the French nomenclature, may not be a position of primary importance.

Availing ourselves, therefore, of the nomenclature in use beyond the Rhine, let us endeavour to become familiar with the topography, in order that we may accurately recognize the seat of the lesions, in the observations recorded by L. Türck.

I place under your observation, a frontal section taken across the cerebral hemispheres, immediately behind the corpora mammillaria

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[1] 'Sitzungsber. der Kais. Akademie der Wissenschaften zu Wien,' 1859. *V. infra*, the analyses of these cases.

(Fig. 18). You recognize on this section, just exterior to the middle ventricles, the *nucleus caudatus* (or intra-ventricular nucleus of the corpus striatum), which, in this region, is merely represented by a very small portion of gray matter,—beneath and interior to it, the *optic thalamus*, here largely developed; external to this lies the *capsula interna*, formed principally by bands of white substance which are simply the prolongations of the lower stage of the crura cerebri; these proceed to expand in the centrum ovale to assist in constituting the corona radiata:—external to this is the *extra-ventricular nucleus of the corpus striatum*, in which you distinguish three secondary nuclei denominated by the numbers 1, 2, 3; the third, or outermost, is sometimes designated by the term *putamen*. Still more external is a thin lamina of white matter, the *capsula externa*, and finally, a small band of gray substance, the rampart (or claustrum) (*Vormauer*).[1]

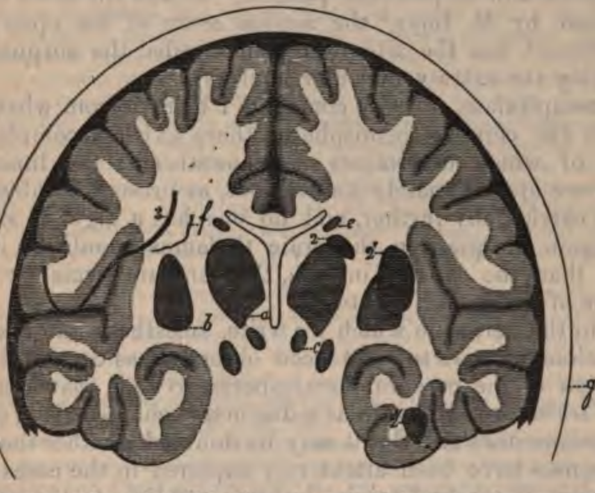

FIG. 18.—Transversal section of brain,—*a*, optic thalamus;—*b*, corpus striatum, lenticular nucleus;—*c*, corpus striatum, caudate nucleus;—*f*, indication of the radiating corona of Reil;—2, 2′, 2″, apoplectic foci (Obs. ii, in 'Türck's Memoir,' *v. infra*, pp. 212–14);—3, vestige of an apoplectic focus (Obs. iii, in 'Türck's Memoir').

Now, gentlemen, in the cases recorded by Herr Türck, the lesions had invaded alike the superior and external portion of the optic thalamus, the third nucleus of the extra-ventricular portion of the corpus striatum, the superior portion of the capsula interna, the corresponding region of the radiating corona, and the adjacent white substance of the posterior lobe.

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[1] The *vormauer* of German anatomists, the *avant-mur*, *rempart*, or *noyau rubané* of the French, is a band of gray matter which, arising from the superior portion of the amygdala, curves round towards the white substance of the convolution bounding the fissure of Sylvius. (S.)

We have consequently complex lesions to deal with here, but they, at all events, allow the region which requires investigation to be circumscribed. Further researches, when sufficiently numerous, will enable us soon to ascertain the fundamental lesion, to which the existence of the hemianæsthesia should be attributed.

Some other cases of hemianæsthesia, of cerebral origin, which have been published since those of Türck appeared, testify to lesions occupying the same circumscribed regions of the encephalon; they make, however, no important additions to the results obtained by that observer. Such amongst others, is the case recorded by Dr. Hughlings Jackson,[1]—here again the alteration was not confined to the *thalamus;* it extended to the extra-ventricular nucleus of the corpus striatum, and consequently the capsula interna must have been affected in its posterior portion. It was the same in the case described by M. Luys,[2] the *median centre* of the optic thalamus was affected, but the alteration had invaded the corpus striatum, (probably the extra-ventricular nucleus).

To recapitulate, we may conclude I believe from what precedes that, in the cerebral hemispheres, there exists a complex region, lesion of which determines hemianæsthesia; the limits of this region are approximately known, but, at present, localization cannot be carried any further, and no one has a right to say that in the region in question, the optic thalamus should be inculpated rather than the capsula interna, the centrum ovale, or the third nucleus of the corpus striatum.

Up to the period in which we write, anæsthesia of general sensibility alone appears to have been observed, as consecutive on an alteration of the cerebral hemispheres, so that *obnubilation of the special senses* would remain as a distinctive characteristic of hysterical hemianæsthesia. But it may be doubted whether the organs of these senses have been attentively explored in the cases of hemianæsthesia of cerebral origin, hitherto published; the records are silent with respect to it.[3]

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[1] The disease was not strictly limited to the thalamus. . . . Outwards the disease extended through the small tongue of corpus striatum which curves round the outside of the thalamus, and thence up to the gray matter of the convolutions of the Sylvian fissure. ('London Hospital Reports', *loc. cit.*, t. iii, p. 376.)

[2] Luys, 'Iconographie photographique des centres nerveux,' p. 16.

[3] At the period when this Lecture was delivered, we were only acquainted with the observations of L. Türck by the brief mention made of them in Rosenthal's 'Treatise on Diseases of the Nervous System.' Since then, thanks to the courtesy of M. Magnan, we have been enabled to procure the complete translation of Türck's memoir (Ueber die Beziechung gewisses Krankheitsherde des grossen Gehirnes zur Anasthesie,' Aus dem xxvi Band, S. 191, des Jahrganges, 1859, des Sitzungsberichte der Mathem. Naturw. Classe der Kais. Akademie der Wissenchaften). We think it useful to give the substance of this work. After recalling

For my own part, I am inclined to believe that the participation of the special senses will be one day recognized, when care shall

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the fact that, usually, in hemiplegia caused by the formation of apoplectic foci in the brain (hemorrhage and ramollissement), the sensibility re-appears very promptly as a general rule, the author relates four cases where, on the contrary, the anæsthesia persisted in a high degree of intensity.

Case 1.—Fr. Amerso, æt. 18. In August, 1858, left hemiplegia, speedy re-appearance of motor power. *12th. Nov.*—The movements of the left upper extremity are rapid and energetic; those of the corresponding inferior extremity exhibit slight paresis. Very intense anæsthesia exists on the left side (limbs, body, etc.). Facial sensibility is diminished, on this side only. Formications from time to time through all the left side. Died, 18th March, 1859.

Autopsy.—At the base of the corona radiata of the right hemisphere, immediately outside of the tail of the corpus striatum, appears a lacuna of the size of a pea (*cellular infiltration*). The anterior wall of this lacuna is two lines behind the anterior extremity of the optic thalamus. Two or three lines farther off, another lacuna is seen, of smaller dimensions, which extends to four or five lines behind the posterior extremity of the thalamus, so that as the usual length of the optic thalamus is eighteen lines, the portion of the corona radiata which lies immediately adjacent to the tail of the corpus striatum was perforated, fore and aft, by the old focus of ramollissement for an extent of eleven lines. A similar focus involves the external portion of the third part of the lenticular nucleus. It commences nearly two lines behind the anterior border of the optic thalamus and ends at about four lines from the exterior extremity of the optic thalamus. In its course of one inch long, it occupied the greater length of the internal side of the third part of the lenticular nucleus, and part of the capsula interna. In the posterior half of their course, then two foci were not farther part, in one place, than the distance of one line. It follows, that in this place, almost all the corona was separated from the internal capsule and the optic thalamus. *Spinal cord.*—Collection of granular bodies somewhat abundant in the left lateral column, rare in the anterior column.

Case 2, S. J., æt. 55.—Apoplectic attack, followed by hemiplegia, Oct. 25, 1851. Two months after, the paralysis of the extremities disappeared to such an extent that the patient could extend the arms, grasp objects with some strength, and walk without help, but lamely. *Oct.*, 1855.—Anæsthesia of the left extremities (face and body also benumbed, but in a less degree) persisting since the attack. Power of motion recovered, but the limbs of the left side are more feeble than those of the right. Died Oct. 31, 1858.

Autopsy.—Old flat cicatrix, about five lines in breadth and eight in length, situated at the superior and external part of the right optic thalamus. The cicatrix begins four and a half lines behind the left anterior extremity of the optic thalamus, and ends eight lines farther off. Lying parallel to this cicatrix is another, an inch long, occupying the third part of the lenticular nucleus; it begins two lines behind the anterior extremity of the thalamus and ends nearly three lines in front of its posterior extremity (fig. 18, 2, and 2′). There was, besides, a lacuna in the right inferior lobe (fig. 18, 2″), another in the anterior lobe of the same side, two as big as a pin's head in the anterior part of the right optic thalamus; two in the pons Varolii, and finally, one in the right and superior portion of the left hemisphere of the cerebellum. No secondary degeneration of the cord was observed.

Case 3.—Fr. Hasvelka, æt. 22. November 1st, 1852. Apoplectic attack, hemiplegia on the right, with intense anæsthesia of the corresponding half of the body. At the end of five weeks, diminution of motor paralysis. *February* 3, 1853.—Motion quite free, on the right side. The entire right half of the body is the seat of very marked anæsthesia (scalp, ear, face, and body). The anæsthesia is equally noticeable in the eyelid, nostril, left half of the lips, and not only on the outer but also on the inner side. The right conjunctiva is less sensitive than the left When the right nostril is tickled, the sensation is less felt than in the left. Same difference between the right meatus auditorius, and the left. In the right half of the mouth (tongue, palate, gums, cheek), the sensation of heat is have been taken to seek for it. My opinion is founded on the following basis:—

There exists in the clinical history of the organic diseases of the nervous centres a symptomatic sign but little known, and little remarked as yet, which I shall have occasion some day to discuss in detail before you. This is a kind of rhythmical convulsion which occupies an entire half of the body, including the face (in many instances at least), and which assumes sometimes the appearance of the clonic jerking of chorea, sometimes that of the tremor of paralysis agitans. This hemilateral trembling occasionally presents itself as a primary affection; at other times it supervenes consecu-

less vivid than in the left. At the tip of the tongue on the right, and over the space of an inch in length, the patient does not feel the *taste* of salt. Same result as regards the right half of the dorsum and root of the tongue. On the right side also, the sense of *smell* is weakened, and *vision* is less distinct. When the pupils have been made to contract by bringing a light close to the eyes, the right pupil afterwards dilates more than the left, the sense of hearing is normal on both sides. *February* 26.—The anæsthesia has diminished; the movements are more energetic. *March* 15.—Temporary improvement of vision: no difference between the two eyes. *April* 3.—The anæsthesia still exists over the right half of the body (on touch, and pinching). Debility of vision augmented on the right. Died April 4.

Autopsy.—In the white substance of the left superior lobe, is found a focus of ramolissement two inches in length and one in breadth. It dipped into the inferior convolutions of the operculum, and attained the surface of the brain. Its posterior extremity corresponded to that of the optic thalamus; its anterior part greatly exceeded that of the thalamus. In its broadest portion, the focus was only separated by three lines from the tail of the corpus striatum. The convolutions lying beneath, were to the extent of a florin, yellow, softened, and depressed. (Fig. 18, 3.) Optic thalamus healthy, perhaps a little fragment of the third part of the lenticular nucleus has been touched. The focus had destroyed a somewhat considerable length of white substance, and the two external thirds of the foot of the corona radiata. *Spinal Cord.*—Slight agglomeration of nuclei in the most posterior part of the lateral column.

Case 4.—Anne B—, an aged woman, died 22d February. For many years she had right hemiplegia, with intense anæsthesia of the same side. In addition, sensorial anæsthesia (sight, smell, taste) of the same side, and formications.

Autopsy.—Old apoplectic focus, pigmented brown, situated along the outer part of the left optic thalamus, and quite close to the tail of the corpus striatum. It commences six lines behind the anterior extremity of the thalamus and extends two or three lines in front of its posterior extremity. Anteriorly, it is half a line, and posteriorly two or three lines beneath the superior surface of the thalamus, which is considerably depressed in this direction. An inch long and four or five lines deep, the focus touches a large extent of the posterior part of the radiation of the crus cerebri, a part of the internal capsule, and, perhaps, a part also of the lenticular nucleus. *Spinal Cord.*—Accumulation of granular bodies in the posterior part of the right lateral column.

To sum up: the foci were seated at the external periphery of the optic thalami, they extended, from before backwards, in the longitudinal axis of the cerebrum, without, in most cases, reaching the extremities of the thalamus. They were from eight lines to an inch in length, reaching even two inches into the white substance. The regions affected were: the superior and external part of the thalamus; the third part of the lenticular nucleus; the posterior part of the internal capsule, comprised between the thalamus and the lenticular nucleus; the corresponding portion of the white substance of the superior lobe opposed to it. Several of these regions were always affected together. The fibres which proceed from the white substance of the hemisphere into the external part of the optic thalamus were constantly affected.

tively on a hemiplegia, whose invasion was sudden. In the latter case, it commences to appear at the epoch when motor paralysis begins to improve. The lesion consists in the presence either of a focus of hemorrhage or of ramolissement, or in that of a tumour. In all cases of this kind, which I have hitherto observed, and in the analogous facts collected from various authors, the lesion in question occupied the posterior region of the ophthalmic thalamus and the adjacent parts of the cerebral hemisphere exterior to it.

Now, hemianæsthesia is a tolerably common—but still not a constant—accompaniment of this group of symptoms, and it occupies the same side of the body as the tremor.[1]

It existed in a high degree of development, in a male patient whose history M. Magnan has recently communicated to the *Société de Biologie;* in his case, the form of tremor of which I have tried to give you a summary notion, showed itself in a most marked manner. Everything tends to show (I cannot be more positive as there was no autopsy) that the encephalic lesion was, in this man, of the same sort, with respect to position, as that which I found in my patient. Now, in this case, M. Magnan ascertained, in the clearest manner, that tactual sensibility was not alone involved; the special senses were themselves affected, as they are in hysterical hemianæsthesia. On the side stricken with hemianæsthesia, the eye was affected with amblyopia, the sense of smell was lost, and taste was completely abolished.

Hence, it becomes probable, if I am not mistaken, that complete hemianæsthesia, with derangements of the special senses,—and consequently, such as is presented in hysteria,—may, in certain cases, be produced by a circumscribed lesion of the cerebral hemispheres.[2]

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[1] See a Lecture of M. Charcot (in 'Le Progrès Médical,' 23 Janvier, and 6 Fevrier, 1875), on 'Hemichorée post-hémiplégique.' (Note to 2d edition.)

[2] The views expressed in this Lecture, relative to hemianæsthesia of cerebral origin, have received further clinical confirmation from the incidents of a case, which we noted, in M. Charcot's wards. (Progrès Médical,' 1873, p. 244), and from the experiments on animals conducted by M. Veyssière (Recherches cliniques et experimentales sur l'hemianesthesie de cause cérébrale,' Paris, 1874). This work also contains some interesting clinical notes. (Note to the 2d edition.)

LECTURE XI.

OVARIAN HYPERÆSTHESIA.

SUMMARY.—Local hysteria of British authors. Ovarian pain; its frequency. Historical remarks. Opinion of M. Briquet.

Characters of ovarian hyperæsthesia. Its exact position. Aura hysterica: first node; globus hystericus, or second node; cephalic phenomena or third node The starting-point of the first node is in the ovary. Lesions of the ovary; desiderata.

Relations between ovarian hyperæsthesia and the other accidents of local hysteria.

Ovarian compression. Its influence on the attacks. Modus operandi. Ovarian compression as a means of arresting or preventing hysterical convulsions known in former times. Its application in hysterical epidemics. Epidemic of St. Médard—the remedy termed "secours." Analogies which exist between the arrest of hysterical convulsions by compression of the ovary, and the arrest of the aura epileptica by ligature of a limb.

Conclusions, from a therapeutical point of view. Clinical observations.

GENTLEMEN,—By the somewhat picturesque and certainly very practical term *local hysteria*, British authors are accustomed to designate most of the accidents which persist, in a more or less permanent manner, in the intervals between the convulsive fits of hysterical patients, and which almost always enable us, on account of the characteristics they present, to recognize the great neurosis for what it really is, even in the absence of convulsions.

Hemianæsthesia, *paralysis*, *contracture*, *fixed painful points* occupying different parts of the body (rachialgia, pleuralgia, clavus hystericus), according to this definition, come under the head of local hysteria.

I.

Among these symptoms there is one which, on account of the predominant part it, in my opinion, plays in the clinical history of certain forms of hysteria, seems to me to deserve your entire attention. I refer to the pain which is felt in one side of the abdomen, especially in the left, but which may occupy both sides, *at the extreme limits of the hypogastric region*. This is the *ovarian pain*, concerning which I said a few words in my last lecture; but I do not wish to make unreserved use of this term before justifying the hypothesis which it implicitly adopts—and this, I trust, will be an easy task.

This pain I shall enable you to touch, as it were, with the finger, in a few moments, and to observe all its characteristics, by introducing to your notice five patients who constitute almost the whole of the hysterical cases, actually existing among the 160 patients who occupy the department devoted in this hospital to women affected by incurable convulsive diseases, and reputedly exempt from mental alienation.

II.

You already perceive, from this simple indication, that iliac pain is a frequent symptom in hysteria; this is a fact long recognized by the majority of observers.

Let it suffice, as regards former times, to mention the names of Lorry and Pujol, who, most particularly, noticed the existence of hypogastric and abdominal pains, in hysterical cases.

It is singular, after this statement, to find that Brodie, who was perhaps the first to recognize all the clinical interest which attaches to local hysteria, does not treat of abdominal pain in a special manner.[1]

It seems as of traditional custom that the practical spirit of English surgeons should be attracted by the clinical difficulties which the local symptoms of hysteria present. Mr. Skey, who in this respect may be regarded as continuing Brodie's work, in a very interesting series of lectures on local or surgical forms of hysteria,[2] as he calls them, expatiates on the iliac pain, or pain of the *ovarian region*, which, in his opinion, is very common, and which, according to his view, but contrary to what really occurs, is chiefly met with in the right side.

You are aware that, in France, Schutzenberger, Piorry, and Négrier, have laid special stress on this symptom, which they unhesitatingly attribute to the abnormal sensibility of the ovary.

In Germany, Romberg has followed Schutzenberger on this topic; however, it is to be remarked that, as regards our contemporaries, most of the German authors pass in almost complete silence over all that relates to this hypogastric pain. This is the case, for instance, with respect to Hasse and Valentiner. Hence, it is clear that this symptom, after having enjoyed a certain degree of favour, doubtless on account of the theoretical considerations connected with it, has gone somewhat out of fashion, at present.

Symptoms, also, as you see, have their destiny: *Habent sua fata.* I should not be surprised if the otherwise very legitimate influence of M. Briquet's work counted for a good deal in the production of this result. It now becomes our duty to examine how far we ought to follow this eminent author in the path which he has marked out for us.

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[1] Brodie, 'Lecture illustrative of certain nervous affections,' 1837.

[2] F. C. Skey, 'Hysteria—Local or Surgical forms of Hysteria,' six lectures, London, 1870.

III.

I am far from saying that M. Briquet did not recognize the very frequent existence of fixed abdominal pains in hysterical cases. He has even coined a new word to designate these pains *cœlialgia* (from κοιλία, the belly), and a word, even though it be merely a word, is still something that arrests the mind. In 200, out of 430 cases of hysteria, M. Briquet met with cœlialgia. However, I should point out to you that, under this name, he includes alike the pains of the upper part of the abdomen and those of the iliac and hypogastric regions, but the latter are confessedly the most common.

At first glance, therefore, it seems as if the difference between M. Briquet and his predecessors were merely an apparent one. Now, it is nothing of the kind, and here is the chasm which divides them.

Whilst MM. Schutzenberger, Piorry, and Négrier place in the ovary the chief seat—the focus, so to speak—of the iliac pain, M. Briquet only admits the existence of a simple muscular pain, an *hysterical myodynia.* According to his view: 1st, pain of the pyramidalis or of the lower extremity of the rectus abdominis has been mistaken for *uterine pain;* 2d, pain of the lower extremity of the obliquus abdominis takes the place of the so-called *ovarian pain.* Such is the thesis of M. Briquet.

IV.

Let us investigate together, gentlemen, the basis on which this opinion rests. In order to arrive at our object, I am about to refer to the observations which I have been enabled to collect upon a large scale in this hospital. I shall, therefore, proceed to describe this pain, such as I have learned to know it.

1st. Sometimes it is an acute, nay, a very acute pain; the patients cannot tolerate the slightest touch, nor suffer the weight of the bedclothes, etc.; they shrink suddenly, and as if instinctively, from the finger of the investigator. Add to this a certain degree of tumefaction of the abdomen, and you have the clinical appearance of *false peritonitis*—the *spurious peritonitis* of British authors. It is manifest that the muscles and the skin itself share in the suffering here. The pain then occupies a considerable extent of the surface of the body, and, consequently, is not easily localized. However, Todd[1] remarks, and I have frequently verified the accuracy of his statement, that in certain cases, a circumscribed cutaneous hyperæsthesia occupies a rounded dermal space of from two to three inches in diameter. This hyperæsthesia has its seat partly in the hypogastrium, partly in the iliac fossa, and corresponds, according to this author, to the region of the ovary.

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[1] Todd, 'Clinical Lectures on the Nervous System.' Lecture xx, p. 448, London, 1856.

2d. In other cases, the pain does not spontaneously show itself; it requires pressure to discover it, and, under such circumstances, we note the following phenomena: *a*, there is a general anæsthesia of the *skin; b*, the *muscles*, if relaxed, may be pinched and raised without causing pain; *c*, this preliminary exploration proves that the seat of the pain is neither in the skin nor in the muscles. It is consequently necessary to push the investigation further, and by penetrating, as it were, into the abdominal cavity by pressure of the fingers we reach the real focus of the pain.

This operation allows us to make certain that the seat of the pain in question is usually fixed, that it is always nearly the same; and indeed, it is not uncommon to find that patients point it out, with perfect unanimity. From a line uniting the anterior superior spines of the ilia, let fall the perpendicular lines which form the lateral limits of the epigastrium, and at the intersection of these vertical and horizontal lines will be found the focus of pain, as indicated by the patient, and which becomes further manifest on pressure being applied by the finger.

Deep exploration of this region allows us readily to recognize part of the superior inlet which describes an inwardly concave curve; this is our guiding point. Towards the middle part of this rigid crest, the hand will usually meet with an ovoid body, elongated transversely, which, when pressed against the bony wall, slips under the fingers. When this body is swollen, as often happens, it may attain the size of an olive, or of a small egg, but with a little experience its presence can be easily ascertained, even when it is of much smaller dimensions.

It is at this, the period of exploration, that the pain is chiefly determined; it then manifests itself with characters which may be called *specific*. This is no common pain we have to do with, but a complex sensation which is accompanied by all, or some, of the phenomena of the *aura hysterica;* such as they spontaneously show themselves before an attack. When the sensation is thus determined, the patients recognize it as familiar—as having felt it scores of times.

In short, gentlemen, we have succeeded in circumscribing the initial focus of the aura, and, by the same act, we have provoked irradiations in the direction of the epigastrium (the *first node* of the aura, to use M. Piorry's terminology) sometimes complicated with nausea and vomiting, then, if the pressure be continued, there soon supervene palpitations of the heart, with extreme frequency of the pulse, and finally, the sensation of the globus hystericus is developed in the throat (*second node*).

At this point terminates the description, given by authors, of the ascending irradiations which constitute the aura hysterica. But, judging from my own observations, the enumeration of symptoms, if thus limited, would be incomplete; for an attentive analysis allows us to ascertain the presence, in most cases, of certain cephalic disorders which are evidently the continuation of the same series of phenomena. Such are, for instance (in case of compression of the left ovary), the intense sibilant sounds in the left ear, which the patients compare to the strident noise produced by the whistle of a railway engine—a sensation as of blows from a hammer falling on the left temporal region—and, lastly, a marked obnubilation of sight in the left eye.

The same phenomena show themselves in the corresponding parts of the right side, when pressure is applied on exploration of the right ovary.

The analysis cannot be carried further, for when matters have arrived at this point, consciousness becomes profoundly affected, and, in their confusion, the patients no longer retain the faculty of describing what they feel. Besides, the convulsive fit soon supervenes, if the experiment be persisted with.

Leaving out of the question the phenomena which relate to the last phase of the aura hysterica (the *cephalic symptoms*), I have just been describing to you, gentlemen, the whole series of phenomena obtained in the experiment of Schutzenberger, and we are thus led to acknowledge, with this eminent observer, that compression of the ovarian region, simply reproduces artificially the series of symptoms that spontaneously present themselves in the natural course of the disorder.

I am well aware that, according to M. Briquet, the aura hysterica starts, in the immense majority of cases, from the epigastric *node;* neither do I forget that, in support of his assertion, this author quotes an imposing array of figures. But we must not always bow to statistics, and it may be fairly asked whether M. Briquet, who has shown himself somewhat severe upon the "ovarists," has not in his turn allowed himself to be carried away by some preoccupation which made him neglect to inscribe the initial iliac pain in the series of phenomena of the aura.

If I am to judge from my own observations, this *iliac pain* always precedes in point of time, however small the interval may be, the epigastric pain, in the development of the aura, and consequently it constitutes the first link of the chain.

V.

It remains for me, gentlemen, to establish that this particular point, where the iliac pain of hysterical patients resides, corresponds exactly with the position of the ovary, then I shall have rendered it highly probable, if not absolutely demonstrated, that the painful oval body, whence the irradiations of spontaneous or provoked hysteria start, is really the ovary itself.

Generally, I believe, an imperfect idea is formed of the precise position which the ovary occupies during life. When, the abdomen being laid open, and the intestines raised, we find in the pelvis behind the uterus, in front of the rectum, the appendages of the uterus flabby, shrunken, and as it were shrivelled, it is plain that we are in presence of appearances not at all answering to vital conditions. It is evident that, after death, the arterial network of the Fallopian tubes and of the ovaries (the vascular wealth and erectile properties of which have been so well illustrated by my friend Professor Rouget, of Montpelier), has long ceased to fulfil its functions. Again, it must not be forgotten that the laying open of the abdomen most certainly alters the true relations of the appendages of the uterus to other parts. This is proved by the fact that, in frozen corpses[1], the ovaries occupy a more elevated position,—one which recalls to some extent their admitted position in the newborn infant. In the diagram before you, which is copied from the 'Atlas' of M. Legendre, you see a horizontal transverse section of the body of a woman, aged 20; its plane passes three-quarters of an inch (2 centimètres) above the pubis, and divides one of the ovaries in twain, whilst the other, lying superior to it, escapes. From this it appears that, in the adult female, the ovary should be situated on a level with or even a little above the superior inlet, (or brim of the pelvis) jutting over into the iliac fossa along with the Fallopian tube. This result accords in every particular with that given by palpation applied to the living body. I will add that if you pass a long needle perpendicularly through a corpse laid on the dissecting table, at a spot corresponding with that where hysterical patients complain of iliac pain, you have every chance—as I have frequently found—of transfixing the ovary.

This position of the ovary appears, in fact, to have been implicitly recognized by Dr. Chéreau in his excellent treatise on diseases of the ovary,[2] when he remarks that, in women, where the abdominal wall does not offer too great a resistance the tumefaction, or even the sensibility only, of the ovary may be ascertained. The introduction of the finger into the rectum would not be a superior mode of exploration, according to our author, except in cases where the abdominal parietes present an invincible obstacle.

Gentlemen, after all these explanations which I have just discussed, I believe I have a right to draw the conclusion that it is to the *ovary*, and the *ovary alone*, we must attribute the *fixed iliac pain of hysterical patients*. It is true, that at certain epochs, and in severe cases, the pain, by a mechanism which I need not at present indicate, extends to the muscles and to the skin itself, so as to justify the description given by M. Briquet; but I cannot too often repeat that, if limited to these external phenomena, the description would be incomplete and the true focus of the pain misapprehended.

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[1] E. Q. Legendre, 'Anatomie Chirurgicale homolographique,' etc., pl. X, Paris, 1858

[2] Chéreau, 'Etudes sur les maladies de l'ovaire,' Paris, 1841.

VI.

This would be the place to investigate what is the anatomical condition of the ovary in cases where it becomes the seat of the iliac pain of hysterical patients. In the actual state of affairs, we can unfortunately only give you some rather vague information, in reference to this subject. There occasionally exists a more or less marked tumefaction of the organ, such as was found in the case of blennorrhagic ovaritis recorded in the memoir of M. Schutzenberger. But this is rather an exceptional circumstance, and it is proper to remark that common inflammation of the ovary may exist with all its characters, and yet there shall supervene no *irradiations*, as described, neither spontaneously nor under the influence of pressure. M. Briquet has not failed to set this circumstance prominently forward, and here he is perfectly right. Hence we must emphatically declare that every *ovarian inflammation* is not indifferently adapted to provoke the development of the aura hysterica. Ovarian tumefaction in hysterical patients is sometimes completely absent, at other times it is but little marked; and it seems probable enough that the tumefaction of the ovary, in such cases, is the result of a vascular turgescence analogous to what is exhibited after the occurrence of certain neuralgic affections. Pathological anatomy has not hitherto supplied us with any positive data in relation to this question; at present, therefore, you may designate the state of the ovary either by the term *hyperkinesis* (Swediaur), or *ovarialgia* (Schutzenberger), or *ovaria* (Négrier)—the name, indeed, matters little, when the fact is well established.

VII.

It being conceded that the ovary is the starting-point of the aura hysterica, at least in a group of cases, it will not be uninteresting now to show that an important and, in some sort, an intimate relation exists between the *ovarian pain* and the other phenomena of local hysteria.

You can in fact discern, gentlemen, in the patients to whom I call your attention a remarkable concord between the seat of the iliac pain and the manner in which the concomitant symptoms are localized. I will not revert to the cephalic phenomena of the aura which, as I stated a little ago, are manifested on the same side with the ovarian pain; I will confine myself to showing that the *hemi-anæsthesia*, the *paresis*, and the *contracture of extremities*, occupy the left side when the *ovaria* is situated on the left, and *vice versâ*. I will also point out to you that when the ovarian pain occupies both left and right sides, the other phenomena become *bilateral*, predominating however on the side where a greater intensity of iliac pain is felt.

On several occasions, we have noted in some of our patients an abrupt change of the seat of ovarian pain. The patient Ler— is one of these. When the ovaria, in her case, predominated in the left side, the cephalic symptoms of the aura, the contracture of extremities, etc., showed, for the time, their maximum of development on the same side—predominating afterwards on the right side, when the right ovary became again the more painful.

It must not be forgotten that ovarialgia appears to be a constant phenomenon, one eminently permanent, in the form of hysteria which engages our attention, so that, taken in connection with some other indication of the same category, it may guide your diagnosis in difficult cases.

VIII.

It remains for me now, gentlemen, to enter upon an exposition of facts which will probably be considered by you as the main feature of this study. These facts, in reality, are of a nature, if I err not, to set out in still greater prominence the truly predominant part pertaining to ovarialgia in *one of the forms of hysteria*.

You have just seen how methodical compression of the ovary can determine the production of the aura, or sometimes even a perfect hysterical seizure. I propose now to show you that a more energetic compression is capable of stopping the development of the attack when beginning, or even of cutting it short when the evolution of the convulsive accidents is more or less advanced. This, at least, is what you can very plainly discern in two of the patients whom I have placed before you. In their cases, the arrest of the convulsion, when compression has been properly applied, is total and final. In the others, the manipulation merely modifies the phenomena of the seizure in varying degrees, without, however, producing complete cessation. And be kind enough to note carefully that we have to deal in all of them, not with common vulgar convulsive hysteria, if I may so express myself, but with convulsive hysteria in what is unanimously recognized as its gravest type—I mean *hystero-epilepsy*.

Let us suppose that one of these women is taken with a seizure. The patient suddenly falls to the ground, with a shrill cry; loss of consciousness is complete. The tetanic rigidity of all her members, which generally inaugurates the scene, is carried to a high degree; the body is forcibly bent backwards, the abdomen is prominent, greatly distended, and very resisting.

The best condition for a perfect demonstration of the effects of ovarian compression, in such a case, is that the patient should be laid horizontally in dorsal decubitus, on the floor, or, if possible, on a mattress.[1] The physician then, kneeling on one knee, presses the closed hand or fist into that iliac fossa, which he had previously learned to regard as the habitual seat of the ovarian pain.

At first, he must throw all his strength into the effort in order to vanquish the rigidity of the abdominal muscles. But, when this is once overcome and the hand feels the resistance offered by the rim of the pelvis, the scene changes and resolution of the convulsive phenomena commences.

The patient soon begins to make numerous and sometimes noisy attempts to swallow; then consciousness returns almost at the same time, and now the woman either moans and weeps, complaining that you are hurting her (as in the case of Marc—) or else she experiences relief, and testifies her gratitude; "Ah! c'est bien! cela fait du bien!" is always the cry of the patient Gen—, under such circumstances.

Whichever happens, the result in short is always the same, and if you but continue the pressure for two, three, or four minutes, you are almost certain to find all the phenomena of the seizure disappear as if by magic. You may, besides, vary the experiment and at your pleasure, by removing the compression and again applying it, you can stop the seizure or allow it to recur as often almost as you like.

When once we have definitely overcome the obstinate resistance which the abdominal parietes always offer at first, it is not necessary to employ all one's strength, and the application of the two first fingers of the hand to the presumed seat of the ovary is sufficient to produce the desired effect. However, the operation, if it require to be prolonged for some minutes, is always rather fatiguing to the physician. I have contemplated modifying the modus operandi.

Perhaps, you might make use of a bag filled with shot, such as M. Lannelongue has employed for a different purpose, or the application of an appropriate bandage might be tried—this is a question to be considered. At present, the assistants in the wards who have been instructed in the method of manipulation described, apply it day by day in the case of those patients to whom it is really beneficial.

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[1] It may not be amiss to remark that, if the seizure occur whilst the patient is reposing, the method of compression described can also be applied without removing her from bed. Applied in this manner, by Professor Charcot, I have been a witness to its instantaneous effect, in the hysterical wards of La Saltpêtrière. (S.)

IX.

It is singular enough, gentlemen, that a method the practice of which is so simple and which, undoubtedly, is capable of rendering real service, should have fallen, as it has fallen in our days, into complete disuse. As I have already intimated, the invention of this process is far from being my own: it may possibly be traced to a very ancient period; it is certain that it dates from a time anterior to the sixteenth century. The following is what I have learned in reference to it, from some researches made rather hurriedly amongst the dustiest, and therefore the least frequented, volumes of my library.

Willis,[1] in the 17th century, in his treatise on convulsive disease, expressed himself as follows: 'It is certain,' he says, 'that the convulsive spasm which comes from the belly is arrested and can be prevented from ascending to the neck and head by a compression of the abdomen, determined by arms being clasped round the body, or by means of bandages drawn very tight.' He states also that he succeeded himself in stopping a fit, by pressing energetically with both hands joined together upon the lower part of the belly. But Mercado[2] (in 1513) had long previously advised the use of *frictions on the abdomen*, with the object of reducing the womb, which he supposed to be displaced, according to the old doctrine.[3] One of his countrymen, Monartès, it seems, went about it in a more determined manner, for he placed a large stone on the patient's belly during the seizure.

It does not appear, however, that this custom prevailed widely; for I do not find it mentioned in Laz. Rivière, nor in F. Hoffmann. Boerhave alone, at the beginning of the 18th century, insists anew upon compression of the abdomen during the hysterical seizure; it should be applied, according to him, by means of a cushion placed under sheets drawn tight, and extending from the false ribs to the crests of the ilia. In this manner, he says, you give almost certain relief to the patient, provided the sensation of the globus hystericus has not yet ascended beyond the diaphragm.[4]

In modern times, Recamier, reviewing this method, which, as you see, was already ancient, placed on the belly of the patient a cushion upon which an assistant took his seat. His example has been but little followed, so far as I am aware, except by Négrier, the Director of the School of Medicine of Angers, whose 'Collection of facts relating to the history of the ovaries and of the hysterical affection of females,' published in 1858, does not, however, appear to have attracted much attention. The process adopted by Négrier is a more methodical one than those employed by his predecessors; in the application of compression, it is the ovary he aims at, 'a strong and broad pressure exerted by means of the hand upon the ovarian region is sufficient in many cases,' remarks Négrier, 'to ward off and completely suppress the convulsive seizure.'

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[1] Willis, 'De Morbis Convulsivis,' t. ii, p. 34.

[2] D. L. Mercatus, 'Opera, tit. iii, De virginum et viduarum affectionibus,' p. 546, Francof, 1620,

[3] Négrier, "Recueil de faits pour servir à l'histoire des ovaires et des affections hystériques de la femme," Angers, 1858, pp. 158, 169.

[4] Van Swieten, 'Comm.,' t. iii, p. 417.

But, let us put aside for awhile the methods of regular medical practice, and see what have been the processes by means of which, in certain celebrated hysterical epidemics, the assistants gave relief to the "convulsionnaires." Among the modes of succour adopted, we find mention made of one very curious custom well worth examining, the original notion of which must, in all probability, have been owing to the suggestion of some "convulsionnaire;" I allude to *compression of the abdomen*. There are, in fact, hysterical patients, who, on experiencing the premonitory pains of the aura, instinctively seek relief in compression of the ovarian region. Such is the case, for instance, as regards one of our patients, named Gen—, whose symptoms have been already discussed. This woman has long been accustomed to arrest the development of a seizure by compressing the left ovary; she generally succeeds when the invasion of the attack does not take place with great rapidity. If she fail in her effort, she calls on the attendants to help her in the operation.

Let us examine a little more closely these incidents of convulsive epidemics, as we find them narrated in history: they supply material for a retrospective study which is not devoid of interest.

The learned Hecker, writing of those who were affected with St. John's Dance,[1] remarks that they frequently complained of great epigastric pain, and requested to have their abdomen compressed by bandages.

But, in reference to this subject, we find the most interesting documents in connection with the epidemic of St. Médard,[2] as it is called. You are aware how this took place when the religious exaltation of the Jansenists, persecuted on account of the Bull Unigenitus, was at its climax. The epidemic, which began at the tomb of Deacon Pâris, who died in 1727, presented two distinct periods.

The first was chiefly remarkable, at least from our point of view, on account of the cure of a certain number of sick persons, amongst whom were several suffering from well attested permanent hysterical contracture.[3] In the second period, predominated convulsions of a more or less singular character, but which, in short, differ in nothing essential from those which characterize hysteria when it assumes an epidemic form. Now, it was at this period that the practice of giving the *secours* (as it was called) made its appearance in the epidemic of St. Médard.

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[1] Hecker, "Danse de St. Jean," Aix-la-Chapelle, 1374. "Epidemie de St. Witt," à Strasbourg, 1437.

[2] Carré de Montgeron, *loc. cit.*

[3] Bourneville and Voulet, "De la contracture hystérique permanente," pp. 7–17, Paris, 1872.

Of what did this succour consist? In most cases methods were employed to cause firm compression of the abdomen, or else violent blows were given it with some instrument. Thus there were: 1st, the succour administered by means of a heavy andiron, with which the abdomen was repeatedly struck; 2d, the succour given by means of a wooden beetle or large pestle, which differs little from the former; 3d, in this case, a man clasped his two fists together and thrust them, with all his might, against the abdomen of the "convulsionnaire," and, the better to succeed, he called other men to assist him; 4th, three, four, or even five persons got upon the body of the sufferer—a "convulsionnaire," called by her co-religionists, Sister Margot, had a particular predilection for this species of succour; 5th, finally, there is a case where long bands were disposed so that they might be drawn tight to left and right, and thus compress the abdomen. These modes of succour, whichever kind was adopted, were always, it appears, followed by great relief.

Hecquet, a physician of the period, declined to see in these convulsions, which others attributed to divine influence, anything but a natural phenomenon,—and so far he was perfectly right. But I cannot share his opinion when, in his work entitled, 'Du Naturalisme des Convulsions,' he maintains that the modes of succour were nothing else than practices suggested by lubricity. For my own part, I do not well see what lubricity could have to do with blows of pestles and andirons administered with extreme violence, although I am far from forgetting what a depraved taste may give birth to, in this affection. I believe it is very much simpler and very much more legitimate to admit that the succour—apart from the amplifications suggested by a love of notoriety—corresponded to an empirical practice, the result of which was to give great relief in cases of hysterical seizures.

X.

You have assuredly remarked, gentlemen, the analogies which exist between this arrest of hysterical or hystero-epileptical convulsions, determined by abdominal compression, and the arrest of convulsions which is sometimes effected by a *ligature of the limb* from which the phenomena of the aura, in such cases, take their rise. This, perhaps, is the place to remind you that a sudden flexure of the foot causes, as M. Brown-Séquard has shown, the immediate cessation of the convulsive tremulation of *spinal epilepsy*, observable in certain cases of myelitis. You are not aware that, in *experimental pathology*, these clinical facts find, to some extent, their explanation. I cannot enter into details at present, let it suffice to remind you that numerous experiments on animals bear testimony to the fact that suspension of reflex excitability of the spinal cord may be caused by irritation of the peripheral nerves. Thus, the experiment of Herzen shows us that, in the case of a decapitated frog (which was consequently placed in an excellent condition to augment to the utmost the reflex excitability of the spinal cord), if the lower portion of the cord be irritated it will be impossible, so long as this excitation subsists, to call into action the excitability of the superior extremities. And, on the other hand, if you tie a ligature tightly round the upper extremities of a frog, similarly decapitated, so long as this ligature remains, the excitation of the inferior extremities will not be followed by reflex movements. This, at least, is what is demonstrated by Lewisson's experiment.

However, although these facts are more easily analyzed they are not, in the actual state of science, more easily explained than the corresponding phenomena observable in man.

XI.

Time presses, and I cannot dwell any longer upon this subject. I should, however, have liked to show you the importance, from a practical point of view, of suppressing severe fits of hysteria, or, at least, of moderating their intensity. But this aspect of the question may be more appropriately illustrated when I shall have described, in another conference, the consequences which follow reiterated fits,—otherwise termed the *hystero-epileptical acme*.[1] I will confine myself, at present, to formulating as follows, one of the conclusions deducible from the present study:—

Energetic compression of the painful ovary has no direct influence over most of the permanent symptoms of hysteria, such as contracture, paralysis, hemianæsthesia, etc.; but it has a frequently decisive effect upon the convulsive attack, the intensity of which it can diminish, and even the cessation of which it may sometimes determine.

XII.

I have, in conclusion, gentlemen, to introduce to your observation the patients whom I have had chiefly in view in the preceding description, and to point out the most salient peculiarities which they present.

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[1] In French, *état-de-mal hystéro-épileptique*. French pathologists employ the term *état* (*status* ἀκμή) to designate that period of a disease when the symptoms having attained their utmost intensity, may remain for some time stationary, as it were. (S.)

CASE I.—Marc—, æt. 23, suffering from hystero-epilepsy since the age of 16. It is not certain to what cause the origin of the disorder should be attributed. However that may be, she presents the following phenomena of local hysteria: *hemianæsthesia*, *ovaria*, and *paresis*, on the left side. She is likewise subject to frequent vomiting, and has had *achromatopsia* of the left eye.

The attacks are preceded by a characteristic aura; the premonitary phenomena start from the left ovary, and the cephalic symptoms are very marked. With respect to the seizures, they are composed of three periods; *a*, tetaniform, epileptiform convulsions, foaming at the mouth;—*b*, (period of contortions) great movements of the body and lower extremities; during this stage, the patient gives utterance to strange words, and seems a prey to moody delirium:—*c*, tears and laughter announcing the end of the attack. In her case, a prompt and absolute cessation of all the phenomena is determined by compression of the left ovary.

CASE II.—Cot—, æt. 21, hysteria began at the age of fifteen. The ill-treatment she had suffered from her father, who was addicted to alcoholic excesses, and her subsequent career as a prostitute, have doubtless exerted a certain etiological influence. The local hysteria is still more marked in this case, than in the former one. We have to note on the right side *hemianæsthesia*, *ovarian pain*, and *permanent contracture* with *tremulation* of the lower extremity.

The attack is heralded by a distinct aura, proceeding from the right ovary, and terminating in very evident cephalic symptoms. The convulsions, which are chiefly tonic, are complicated by epileptiform phenomena: C— bites her tongue, foams at the mouth, etc. The period of contortions follows, and is very intense. The attack frequently terminates by contortions of the pelvis, with laryngeal constriction, tears, and an abundant flow of urine. In her case, compression of the ovary modifies the intensity of the fit, without, however, arresting it. In the first months of the year, this patient was stricken with the *hystero-epileptical acme*, to which we will return in another lecture.[1]

CASE III.—Legr— Geneviève, was born at Loudun. The coincidence is curious—for that, you know, was the scene of the sad drama of which Urbain Grandier was the victim. Geneviève is 28 years of age; her hysteria dates from the period of puberty. Among the permanent symptoms of local hysteria, we observe well-marked *left hemianæsthesia*, *left ovarian pain*, with easily discerned tumefaction. Her mind, finally, is in a strange state.

The *aura* is very distinct, and the cardiac palpitations and cephalic phenomena constitute the predominant symptoms. Each seizure is divisible into three stages: 1st, epileptiform convulsions, foaming, and stertor; 2d, then contortions of the limbs and entire body; 3d, the period of delirium, during which, at the close of complete attacks, she relates all the incidents of her life.

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[1] This case is detailed in full in the treatise by Bourneville and Voulet, 'De la contracture permanente.' Obs. viii, p. 41.

Sometimes the patient, in this latter phase, has hallucinations; she sees ravens and serpents; moreover, she commences a kind of dance, and then she exhibits, as it were, in an embryonic state and sporadic form, a specimen of those dances of the middle ages, described under the name of *saltatory epidemics* (or the dancing mania). In connection with this, I would have you note that certain cases of hysteria—forming, in some sort, varieties within the species—present in a rudimentary state the different convulsive forms which are exhibited in a highly developed state in the time of epidemics. This, indeed, is a point which Valentiner has thoroughly discussed in his interesting work on hysteria.[1]

In Geneviève's case, compression of the ovary determines what we might call a sudden arrest of the attack. She has a clear conception of its influence, for she herself tries to compress the region which gives birth to the aura, and when she cannot succeed, she, as we have already mentioned, calls for help from the attendants.

Case IV.—Ler—, æt. 48, is a patient well known to all the physicians who, during the last twenty years, have frequented this hospital, in the discharge of various duties. In other words, hers is a *cas célèbre* in the annals of hystero-epilepsy. You will find the early portion of her history narrated in the thesis of M. Dunant (of Geneva). Ler— ceased to menstruate four years ago, notwithstanding which, the nervous accidents persist. We called on you just now to observe the presence of a rudimentary form of *Tarantism* in Geneviève's case; Ler— is a *Demoniac*, one "possessed by a devil,"—or, again, she presents the image, not much fainter than the reality, of one of those women who were called "Jerkers" in the Methodist Camp Meetings, and who assumed the most horrifying attitudes in their paroxysms. (See *Figs.* 19, 20, and 21.)

The probable cause of these nervous accidents in the case of Ler— deserves mention. She had, as she says, a series of frights: 1st, at the age of eleven, she was terrified by a mad dog; 2d, at the age of sixteen, she was struck with horror at the sight of the corpse of a murdered woman; 3d, at the same age, she was again terrified by robbers, who, as she was passing through a wood, rushed out to despoil her of the money she carried.

The components of local hysteria, in her case, are formed by *heminæsthesia*, *ovaria*, *paresis*, and momentary *contractures* of the upper and lower extremities on the right side. Sometimes the phenomena invade the left side, and then, in accordance with our description, we find double ovaria, with double anæsthesia, etc.

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[1] Valentiner (Th.), "Die Hysterie und ihre Heilung," v. q. extract in 'Mouvement Médical,' June, 1872.

The attacks, which are heralded by a well marked ovarian *aura*, are characterized in the first stage, by epileptiform and tetaniform convulsions; after this come great gesticulations of a voluntary character, in which the patient, assuming the most frightful postures, reminds one of the attitudes which history assigns to the demoniacs (period of contortions, Figs. 19, 20 and 21).

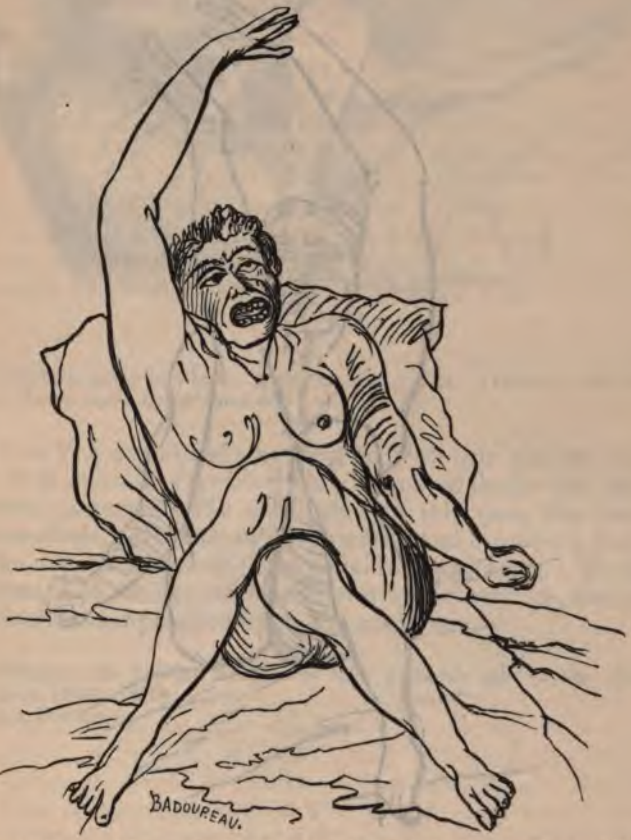

FIG 19—Attitude of Ler— during the attack; period of contortion. (Fac-simile of a sketch from nature)

At this stage of the attack, she is a prey to delirium, and raves evidently of the events which seem to have determined her first seizures. She hurls furious invectives against imaginary individuals, crying out, " villains! robbers! brigands! fire! fire! Oh, the dogs! I'm bitten!"—Reminiscences, doubtless, of the emotions experienced in her youth.

When the convulsive portion of the attack is ended, there supervene, as a general rule: 1st, hallucination of vision; the patient beholds horrible animals, skeletons, and spectres; 2d, paralysis of the bladder; 3d, paralysis of the pharynx; 4th, and lastly, a more or less marked permanent contracture of the tongue.

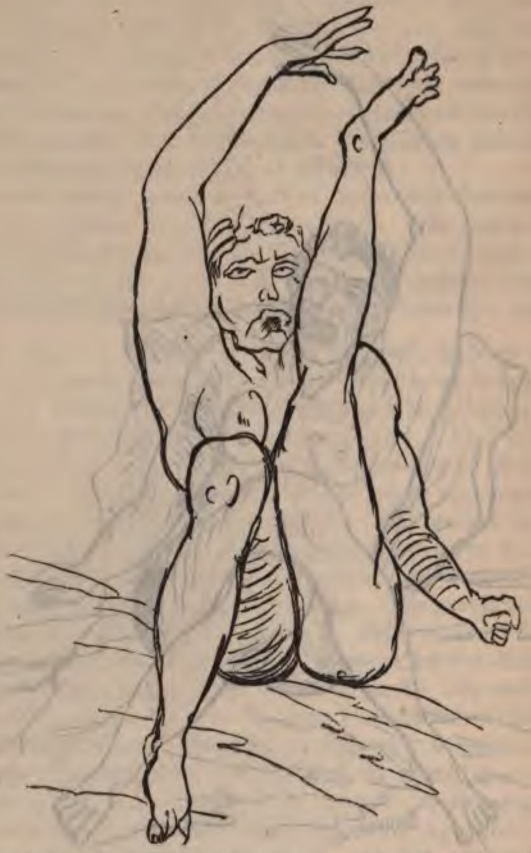

Fig. 20.—Attitude of Ler— during the attack; period of contortion. (Fac-simile of a sketch from nature.)

These latter phenomena occasionally render it necessary to have recourse, for several days, to the use of the catheter, and to the employment of the stomach pump for alimentary purposes.

Compression of the ovary, in this case of Ler—, is almost void of effect upon the convulsions.[1]

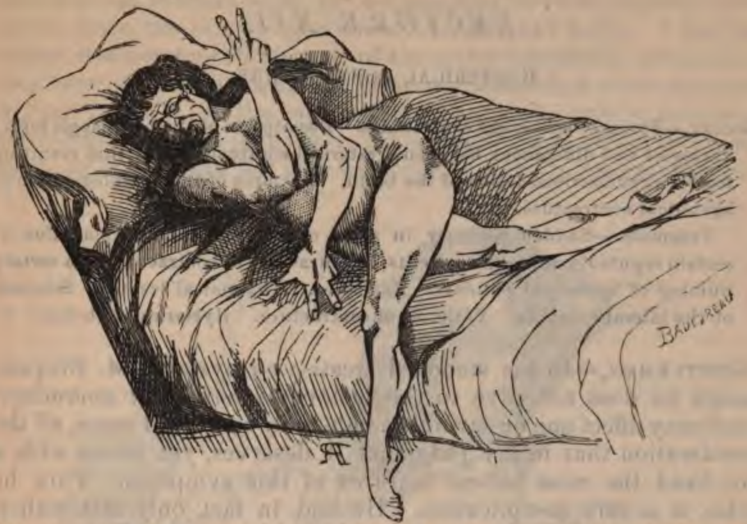

FIG. 21.—Hystero-epileptic attack; period of contortions. (Drawn by M. Richer from a sketch made by M. Charcot.)

CASE V.—You are already acquainted with this patient, named Etch—; it is she who furnished us with materials for our lecture on *hysterical ischuria*.[2] We note, also, in this case, the absence of *hemianœsthesia*, *achromatopsia*, *contracture*, and *ovaria* on the left side. The attacks are principally tetaniform and tonic. We have not, hitherto, had the opportunity of testing, in her case, the influence of ovarian compression upon the convulsions.

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[1] We published the detailed account of this patient's case in the 'Progrès Médical,' (Nos. 18–33, 1875).

[2] *Vide ante*, Lecture ix.

LECTURE XII.

HYSTERICAL CONTRACTURE.

SUMMARY.—Forms of hysterical contracture. Description of the hemiplegic form; analogies and differences between hysterical contracture and that resulting from a circumscribed lesion of the brain. Example of the paraplegic form of hysterical contracture.

Prognosis.—Sudden recovery in some cases. Scientific explanation of certain reputedly miraculous events. Incurability of contracture in a certain number of hysterical patients. Examples. Anatomical lesions. Sclerosis of the lateral columns. Varieties of contracture. Hysterical club-foot.

GENTLEMEN,—In his standard treatise on hysteria, M. Briquet, though he does not give to the history of *permanent contracture*, which may affect one or several members in hysterical cases, all the consideration that in my judgment it deserves, yet traces with a sure hand the most salient features of this symptom. This, he writes, is a rare complication. He had, in fact, only met with it six times at the period when he published his work. In one case, the contracture occupied one limb only; in two others, it appeared under a *hemiplegic form*; and in the last three cases, it assumed the *paraplegic form*. It is perfectly true that hysterical contracture can present these several aspects. You will, besides, verify the fact for yourselves, as I am fortunate enough to be able to place under your observation two patients, one of whom presents the hemiplegic form and the other the paraplegic form of hysterical contracture. We are thus enabled to make you touch with the finger, as it were, the most interesting peculiarities connected with this singular manifestation of hysteria.

I.

Etch—, now aged 40 years, has been affected for twenty months with left hemiplegia. You perceive the *superior extremity* of this side is in semi-flexion (fig. 22); it is the seat of considerable rigidity, a fact attested by the difficulty experienced in trying to increase the flexion, and the impossibility of obtaining complete extension.[1]

The left *lower extremity* is in extension; its several parts are, so to speak, in a forced posture. Thus the thigh is strongly extended on the pelvis, the leg upon the thigh. The foot presents a most marked example of *talipes equino-varus.* In addition to this, the adductor muscles of the thigh are, also, spasmodically contractured. In short, all the joints are alike rigid, and the whole limb forms as it were an inflexible bar, for, by grasping the foot, you can raise, in one piece, the inferior portion of the patient's body. I lay particular stress upon this attitude of the lower extremity, because it is very rare in hemiplegia arising from circumscribed cerebral lesion (*en foyer*), and is, on the contrary, as it were, the rule in hysterical contracture. In the latter case, permanent flexion of the thigh and leg is, to judge from my own experience, a really exceptional fact.

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[1] At the present moment (July, 1873) the contracture of the left extremities is observable, with all the characteristics described in the lecture, which was delivered in June, 1870. [Etch— has since recovered, see *infrà*, pp. 240–1. S.]

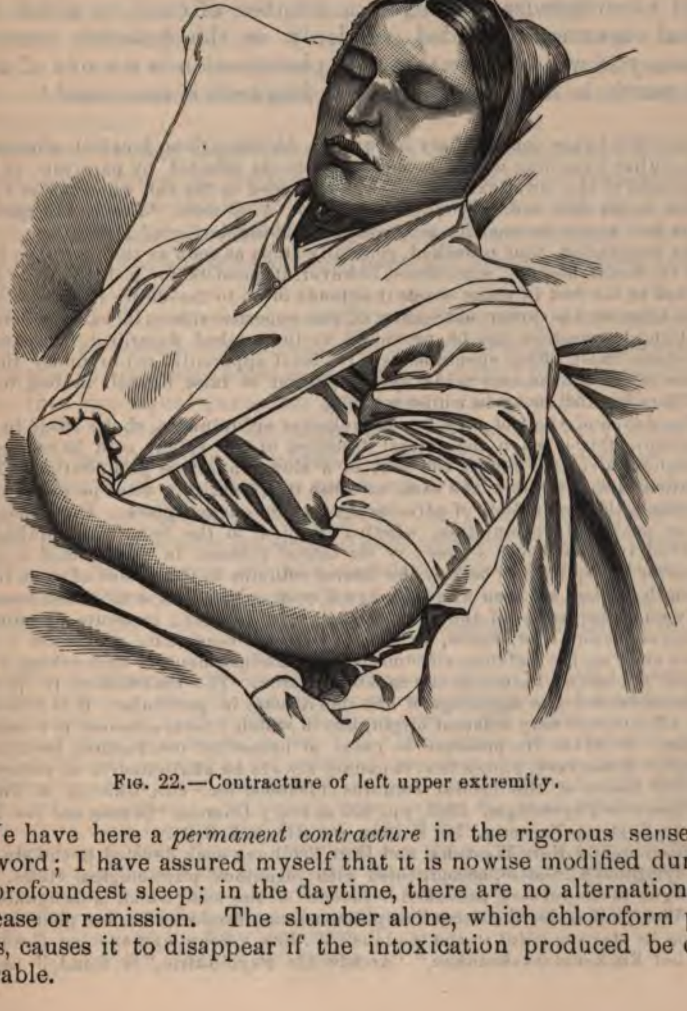

FIG. 22.—Contracture of left upper extremity.

We have here a *permanent contracture* in the rigorous sense of the word; I have assured myself that it is nowise modified during the profoundest sleep; in the daytime, there are no alternations of increase or remission. The slumber alone, which chloroform procures, causes it to disappear if the intoxication produced be considerable.

Although the hemiplegic contracture by which our patient is affected is, I repeat, of nearly two years' standing, you perceive that the nutrition of the muscles has not sensibly suffered. I should also add that the electrical contractility has remained nearly normal.

I would point out to you, in passing, that, by forcibly setting back the point of the foot, you determine in the contractured lower extremity a *trepidation* which sometimes persists long, even when the foot is let go, and allowed to resume its former attitude. You are aware that this same trepidation is very commonly met with in paralysis with contracture, arising from an organic spinal lesion, as, for instance, when the lateral columns are affected with sclerosis; but I have likewise seen it, in a number of cases, in which hysterical contracture ended suddenly in the patient's recovery. Hence, you will observe that this phenomenon is not one of absolute worth, so far as an anatomical diagnosis is concerned.[1]

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[1] In 1868, in the course of my lectures at La Salpêtrière, I called attention to the peculiar trembling which, in certain patients affected by paralysis, or even by paresis, of the lower extremities, was produced in the foot when it was caught by the point and suddenly turned back (*v.* A. Dubois, "Etude sur quelques points de l'ataxie locomotrice progressive," 'Thèse de Paris,' 1868).

The trepidation, thus provoked, generally stops as soon as the foot is no longer kept in dorsal flexion; sometimes, however, it persists for a little time after. Limited to the foot in many cases, it extends often to the entire limb, and sometimes even to the lower extremity of the opposite side. In cases where the trembling in question can be provoked by the method described, it frequently also shows itself, either spontaneously (at least apparently so) or under the influence of the movements made by the patient to raise himself in bed, to rise from it and stand, or again whilst walking.

Trepidation of the foot, whether provoked or spontaneous, shows itself in various circumstances when the lateral fascicles of the spinal cord have become, throughout a certain extent, the seat of a slow connective proliferation. These conditions are, it is plain, the same as those in which, at a later period than the trembling, the production of *permanent contracture* takes place. Thus spontaneous or provoked tremulation, whether limited to the foot or generalized, is observed in *symmetrical sclerosis of the lateral columns*, in *disseminated sclerosis*, whenever the spinal foci occupy the lateral columns to the extent of some inches in length. They are seen when *sclerosis descendens* has been established consecutively on compression of the cord, caused by a tumour; in acute or subacute *transverse myelitis;* or, again, in *lateral sclerosis, consecutive on certain cerebral lesions* such as, for instance, circumscribed ramollissement, or hemorrhage of the opto-striate bodies, involving the capsula interna. The tremulation in question is, therefore, not the appanage of any one disease in particular; it is connected with affections of very different origin, but in which lateral sclerosis is a common feature. However, its presence in cases of hysterical contracture, terminating abruptly in recovery, shows that it cannot always be attributed to a perceptible material lesion of the lateral columns (Dubois, *loc. cit.*, Charcot et Joffroy, 'Archives de Physiologie,' 1869, pp. 632 et seq.; Charcot, 'Leçons sur les Maladies du Système Nerveux,' 1re Edition, pp. 218, 307, 319).

Quite recently Herr Westphal and Herr Erb have each devoted to the study of this symptom a treatise, accompanied with ingenious physiological views. According to these authors, provoked *trepidation* of the foot (which is designated by Herr Westphal under the name Füszphänomenon) would be a reflex phenomenon, having its starting point in the tendons (W. Erb, "Sehnenreflexe bei Gesünden und bei Rückenmarkskranken," 'Archiv für Psychiatrie,' iv Band, 3 heft, p.

Leaving out of the question the difference we have mentioned respecting the attitude of the lower extremity, all the other peculiarities we have described might, strictly speaking, apply to a case of organic hemiplegia, resulting from a deep-seated lesion of the encephalon, as hemorrhage or ramollissement, for instance.

Another feature of resemblance is the following: hemiplegia showed itself suddenly in Etch— during a seizure. After this attack, the patient remained for several days without consciousness.

Having indicated the analogies, I must point out the differences. They are numerous and emphatic, and in point of fact, nothing is more simple than to assign hysterical contracture to its proper cause, by taking note of characters which are almost always present.

1st. Remark, in the first place, gentlemen, the absence of facial paralysis and of deviation of the tongue, when that organ is protruded. You know that these phenomena always, on the contrary, exist to some extent in hemiplegia resulting from circumscribed cerebral lesion.[1]

2d. Observe also the existence of an analgesia and of an anæsthesia, which may be termed absolute, extending over the entire paralyzed half of the body, and consequently occupying the face, trunk, etc. This alteration of sensibility involves not only the skin, but also the muscles, and perhaps the bones; it stops exactly at the median line.

This kind of generalized anæsthesia, occupying a complete half of the body—head, trunk, and members—this quasi-geometrical limitation of the anæsthetic portion by a vertical plane dividing the body into two equal parts, are, as it were, the peculiar property of hysteria.[2] However it happens, this symptom is very rarely observed in hemiplegia of cerebral origin, and in case of spinal hemiplegia, that is, of hemiplegia resulting from a unilateral lesion of the spinal cord, the anæsthesia, as M. Brown-Séquard has shown, would occupy the side opposed to that affected by motor paralysis.

3d. We have many other distinctive characters to point out. The patient is intelligent, and we have no reason to suspect her sincerity. She can, therefore, give us authentic information with

792, 1875; C. Westphal, "Ueber einige Bewegungs-Erscheimungen an gelähmten Gliedern," Idem, p. 883; W. Erb, "Ueber einen wenigbekannten Spinalen Symptomencomplex," in 'Berliner Klin. Wochenschrift,' 1875, No. 26).

In some cases of paralysis of the upper extremities, when, for example, we have hemiplegia consecutive on lesion of the internal capsule, and when permanent contracture is not too marked, we can succeed, by suddenly extending the fingers, in producing a spasmodic trembling of the hand, similar in every respect to *provoked trepidation of the foot.* (J. M. C.)

[1] According to Herr Hasse ('Handbuch der Path.,' etc., 2 Auflag, Erlangen, 1869) Herr Althaus was the first to point out the absence of facial paralysis, and of lingual and buccal deviation in hysterical hemiplegia. This is not the case; the character in question had been, previously, prominently set forth in R. B. Todd's "Lectures on the Nervous System."

[2] V. *ante*, Lecture X on "Hemianæsthesia."

respect to the mode of evolution of her affection. The following is a succinct account of her history.

There were not, it appears, any hysterical antecedents in her case. The disease set in, when she was 34 years of age, after a violent moral shock, with a seizure accompanied by loss of consciousness. This attack, according to all probability, assumed the epileptic form of hysteria. Etch—, in fact, fell during the fit into the fire, and she bears on her face the traces of the burn which she then received. Renewed attacks, at times plainly hysterical, at times exhibiting some of the aspects of epilepsy, supervened, repeatedly, during the following years; but, at the age of 40, appeared the permanent symptoms of hysteria which we have at present to study. We should, therefore, mention in what concurrence of circumstances they were developed, for we shall find there some characteristic features.

a. Menstruation which, until then, had been regular, became disordered; the patient, from time to time, had vomitings of blood;[1] there was considerable tympanites, with acute pain on pressure in the left ovarian region. This pain was of a special character, being accompanied by peculiar sensations which radiated towards the epigastrium, and which were noticed by the patient as heralding most of her seizures. These symptoms, including the tympanites, and retention of urine, are still in existence.

b. Almost simultaneously with the occurrence of these phenomena, Etch— became subject to persistent *retention of urine*, which necessitates the constant employment of the catheter.

c. Matters were still in this state, when, in October, 1868, there supervened a very severe attack, accompanied by convulsions and followed by an apoplectiform condition with stertorous breathing; then *hemiplegia* suddenly made its appearance.

Now, gentlemen, this *considerable tympanites*, these *pains in the ovarian region*, this *retention of urine*, constitute a group of symptoms the importance of which is nearly decisive in diagnosis. Nothing similar is to be seen in the premonitory symptoms of hemiplegia arising from cerebral lesions, whilst it is very common, on the contrary, to find these symptoms preceding the appearance of the permanent phenomena of hysteria, whether hemiplegia or paraplegia. This is a point which M. Briquet has not failed to bring out; it is likewise properly noticed, so far at least as hysterical paraplegia is concerned, by Dr. Laycock, in the following terms: "In hysteria, more or less severe paralysis of the lower extremities is always accompanied," he might have added, "and preceded," "by a corresponding degree of perturbation in the functions of the pelvic viscera; this perturbation is manifested by constipation, tympanites, vesical paralysis, increase or diminution of the urinary secretion, ovarian or uterine irritation, etc."[1]

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[1] This is a frequent accident in hysterical patients, when there is a notable derangement of the catamenia.

d. When Etch— was admitted a year ago (June, 1869) to La Salpêtrière, the hemiplegia had been seven or eight months in existence. Independently of the characteristic peculiarities, already mentioned, the state of the paralyzed members could be, itself, quoted in favour of the hysterical origin of the paralysis. Thus, whilst the upper extremity was in a state of complete and absolute flaccidity, the lower extremity presented a very marked rigidity of the knee. This would be a considerable anomaly in a case of hemiplegia, consecutive on cerebral lesion; for, in such a case, the slowly ensuing rigidity prefers to manifest itself in the upper extremity.

e. The contracture which at present occupies the upper extremity, only dates from a few months back, and it was developed suddenly, and without transition, after a seizure. It is not in this way, as you know, that we find the tardy contracture supervening, which results from hemorrhage or ramollissement of the brain. In the latter case, contracture always sets in slowly and in a progressive manner.

Thus, gentlemen, by taking note of all the circumstances which have just been enumerated, nothing is more easy than to ascertain the real cause of the disease in the case of our patient Etch—. In the following observation, which relates to a case of hysterical paraplegia,[2] the same facilities for making a differential diagnosis may be found.

II.

Alb—, aged 21 years, a foundling, has been affected for about two years with permanent contracture of the inferior extremities, which are, as you may observe, in extension and quite rigid. As in the case of Etch—, muscular contractility is not diminished. The members are emaciated, but this emaciation affects them generally, and is due to the fact that the patient is subject to almost uncontrollable vomitings, which hinder her from taking sufficient nutriment. We have likewise to note an almost complete analgesia of the paralyzed members.

Now, the following are the thoroughly decisive symptoms which allow us to establish the diagnosis.

a. Alb— has been subject to hysterical fits since she was sixteen years of age; *b*, she has been for four years affected with retention of urine, which generally requires the employment of the catheter; *c*, she presents enormous tympanitic distension of the abdomen; *d*, the ovarian regions are painful on pressure, and if the exploration be pressed, an hysterical seizure is soon provoked; *e*, contracture of the inferior extremities supervened suddenly, without transition, and this is a symptom which we have already had occasion to emphasize in the preceding case. Now, such symptoms are not to be met with during the progress of sclerosis of the lateral columns.

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[1] 'Treatise on the Nervous Diseases of Women,' London, 1840, p. 240.

[2] This case was already referred to in Lecture XI. A detailed account of the symptoms may be found in the memoir, 'Comte-rendu des observations recueillies à la Salpêtrière, concernant l'épilepsie et l'hystéro-épilepsie.' (B.)

III.

Thus, gentlemen, nothing, I repeat, is simpler than the clinical interpretation of these two cases, so far as the diagnosis is concerned. But here is a point where, in these and in analogous cases, serious difficulties may arise.

What will become of these patients? In their case, paralysis with contracture has persisted, without amendment, for four years. Will this contracture some day be resolved, or will it, on the contrary, persist indefinitely, and so become an incurable infirmity? These are questions which we must ask, without, however, pledging ourselves to give categorical answers.

A. It is possible that, in spite of its long duration, this contracture may, without leaving any trace of its existence, disappear—perhaps to-morrow, or in a few days, or a year hence. We can foretell nothing concerning it. *In any case, if recovery takes place, it may be sudden.*[1] From one day to the next, resumption of the

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[1] Dr. Laycock remarks that a woman may have been bed-ridden for several months, and quite unable to use her lower extremities, the physician may have given up all hope of being of any assistance to her, when suddenly, under the influence of some potent moral cause, she will be seen to rise from her bed "no longer the victim of nerves, but the vanquisher," as Thomas Carlyle says, and walk about as well as if she had never been stricken with paraplegia. This is one of the terminations of hysterical paraplegia which the physician should never lose sight of, and which well shows what risk he runs in pronouncing a case of this kind to be incurable. T. Laycock, 'A Treatise on the Nervous Diseases of Women,' London, 1840, p. 289. (Note to first French Edition.)

This anticipation was fulfilled during the present year, as regards the first mentioned of the two patients to whom allusion was made in this passage, italicized in the first edition. The state of Etch— on the 21st of May, may be thus summed up: retention of urine, with periodic ischuria, during nine years; contracture of the right lower extremity; contracture of the members on the left side, of six years' standing: contracture of the jaws, necessitating the use of the stomach-pump, of one year's standing; aphonia, lasting during ten months. On the 22d May, at a quarter past seven o'clock in the evening, she was seized with a fit, marked chiefly by great oppression: contracture of the neck-muscles, on the left, which twisted the chin behind the left shoulder. The patient does not lose consciousness, she believes she is going to die; she shrieks—the contracture of the jaws vanishes. She tosses about, the attendants endeavour to restrain her; with her right arm, which has become free, she repels those who hold her. She wants to go to the window for air; and, being opposed, her passion increases, and under this influence it was observed that contracture of the right leg disappeared, and that this was followed by disappearance of that of the left leg, and next by that of the left arm, in succession. Etch— is allowed to rise; she walks about; *in eighteen hours recovery was complete*, or nearly so. Dating from the next day, the urinary secretion became normal again. The amblyopia and anæsthesia normal state may occur; and if it should happen, that at this period the hysterical diathesis is exhausted, the patients may once more take their place in everyday life.

In connection with this, gentlemen, I cannot resist pausing a moment in presence of these rapid and often unhoped-for recoveries from a disease which, during such a length of time, had made itself remarkable on account of its tenacity and its resistance to all therapeutic agents. A sudden strong emotion, a concurrence of events taking powerful hold of the imagination, the reappearance of long-suppressed catamenia, etc.—occurrences such as these are frequently the occasions of those prompt recoveries.

I have seen in this hospital, three cases of the kind which I request your permission to briefly summarize.

1st. In the first case, there was contracture of a lower extremity (fig. 23), of at least four years' standing. On account of the misconduct of this patient, I was obliged to give her a stern admonition and declare that I should turn her out of the hospital. Next day, the contracture had entirely disappeared. This fact is the more important, because convulsive hysteria existed only as a bye-gone fact in her memory. For two or three years past, the contracture had been the only manifestation of the great neurosis.

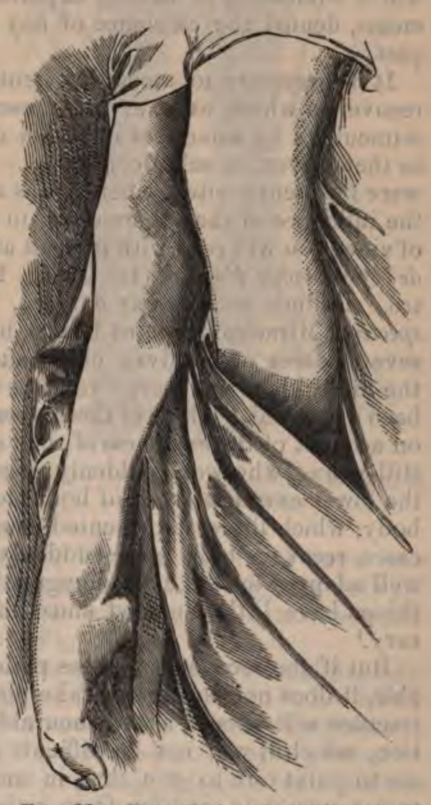

FIG. 23.—Hysterical contracture of the right lower extremity.

2d. The second case, likewise, concerns a woman affected by a permanent contracture of one member only. The hysterical crises, proper, had long disappeared. This woman was

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did not completely disappear until a few days had elapsed, and the patient has only retained some cracking sound in her joints, principally in those of the left leg, as vestiges of her permanent contracture. In conclusion, the only traces of former accidents to-day, are some slight cracking sounds in the joints of the limbs previously affected by contracture. (B.) (Note to the Second French Edition.)

charged with theft; the contracture, which had lasted for two years, vanished suddenly on account of the moral shock caused by this accusation.

3d. In the third case, the contracture had assumed the hemiplegic form; it affected the right side, and was particularly evident in the upper extremity. Recovery took place almost suddenly, eighteen months after invasion, on account of a sudden disappointment. At that time there was no anæsthesia, and the patient, whilst confessing to having experienced strange nervous derangements, denied the existence of any real hysterical seizures in the past.

It is necessary to recognize, gentlemen, the possibility of those recoveries which, even at the present day, have been cried up as miraculous by some, but of which only charlatans take the credit to themselves, in self-glorification. In former times, similar cases were frequently cited, when it was sought to prove before sceptics the influence of the supernatural in therapeutics. From this point of view, you will read with interest an article published in the *Revue de Philosophie Positive* (1er Avril, 1869), by the venerable M. Littré. I allude to an essay entitled, *Un fragment de Médecine rétrospective* (Miracles de Saint Louis), in which is found an account of several cases of paralysis cured after pilgrimages to St. Denis, to the tomb where the mortal remains of King Louis IX had recently been deposited. Three of these cases are especially interesting to us on account of the exactness of their details. They relate to women, still young, who were suddenly seized with contracture of one of the lower extremities, or of both members on the same side of the body, which likewise presented considerable anæsthesia. In these cases, recovery took place suddenly, in the midst of circumstances well adapted to strike the imagination. You see, gentlemen, that things have little changed since the close of the thirteenth century.[1]

But if the recovery of these patients is possible, and even probable, it does not necessarily take place, and it may be that the contracture will persist, as an incurable infirmity. This is an assertion, which it will not be difficult for me to justify. But, allow me to point out to you that, in most authors, you will only find vague, uncertain, and far from satisfactory assertions in reference to this subject.

a. I introduce to you a patient, now aged 55 years, who, eighteen years ago, was seized, after an hysterical attack, with paraplegia accompanied by contracture, the principal characters of which you can still recognize. At the beginning, the contracture from time to time gave evidence of temporary amendment. But, for over sixteen years, it has never undergone the least modification. In this case, we have a real rigidity of the muscles, with predominance of the action of the extensors and adductors. Even after sixteen years of immobility of the lower extremities, the ligamentous parts are not affected, at least not in the knees, as we have been enabled to verify by an exploration made when the patient was under chloroform. The deformity of the feet alone, which resembles that of talipes equino-varus, was not modified during this artificial sleep. The muscles of the legs and thighs are markedly atrophied, and faradic contractility is diminished there. During many years, hysteria seems to have been completely exhausted in this woman, and it has become very improbable that any event could henceforth alter, in any way, the state of her lower extremities. (Fig. 24.)[1]

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[1] Very little, in reality, for the professedly miraculous cures, concerning which so much noise has been made in these later days, do not differ in any perceptible character from the miracles of Saint Louis. The reader may convince himself of this by a perusal of the work recently published by M. Diday, entitled, 'Examen Médical des Miracles,' etc., Paris, 1873 (Bourneville).

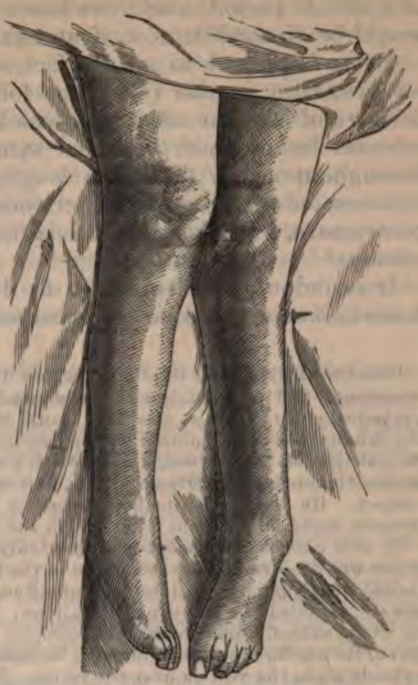

FIG. 24.—Hysterical contracture of both inferior extremities.

b. What condition then has supervened thus to maintain the existence of this paraplegia with rigidity of the limbs? Evidently, in recent cases of hysterical contracture, the organic modification which produces permanent rigidity, whatever it may be, whatever seat it occupy, is very slight, and very fugitive, since its correlated symptoms may disappear suddenly and without transition. It is certain that, with the means of investigation which we possess at the present day, the most minute necroscopic scrutiny would not be capable of discovering, in such cases, the traces of this alteration. But is it the same with respect to inveterate cases? No, gentlemen, I believe I can assert, basing my opinion on my knowledge of an analogous case, that in this woman there supervened, at a certain period, a sclerous lesion of the lateral columns, which would be discernible now, if an examination were possible.

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[1] For a detailed account of this case see p. 53 of the memoir entitled 'De la contracture permanente,' etc. (B.)

It has happened to me, in fact, once to discover, in the case of an hysterical woman (who was for ten years affected with contracture of all four members, which had suddenly supervened), a sclerous lesion which occupied symmetrically the lateral columns throughout nearly the whole length of the spinal cord. On several occasions this woman experienced temporary remissions of the contracture, but after a last seizure, it had become definitely permanent.[1]

It is undoubtedly legitimate to draw from the foregoing facts[2] some inductions relative to the pathological physiology of hysterical contracture. According to the considerations we have mentioned, the lateral columns, or at least their posterior portions—which preside over permanent contracture in cases of disseminated or fasciculated sclerosis—are indicated as being the seat of organic modifications, which are at first of a temporary character, and give rise to hysterical contracture. In the course of time these modifications, whatever they may be, give place to deeper material alterations,—genuine sclerosis is established. This may not be, perhaps, beyond the resources of our art, but, in any case, its existence most assuredly no longer allows us to hope for that sudden disappearance of contracture which forms one of the most striking characters of the disease, when it has not as yet reached the most advanced phases of its evolution.

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[1] Société Médicale des Hôpitaux, Séance du 25 Janvier, 1865. Precisely as we, sometimes, find a spinal lesion, anatomically perceptible, in inveterate cases of hysterical contracture, so also may visual troubles be accompanied by lesions of the fundus, which an ophthalmoscopic examination will reveal. A student of La Saltpêtrière, M. A. Svynos, has given in his inaugural thesis ('Des Amblyopies et des Amauroses Hystériques,' Paris, Julliet, 1873) nearly all that relates to this subject. He has, in particular, described in detail the ophthalmoscopic phenomena noted on different occasions in the case of Etch—.

In this case, which has been repeatedly referred to (Lecture IX and XI), no lesion was discovered for a long time in the fundus of the left eye, affected by hysterical amblyopia; but a later examination, made March 20th, 1873, by M. Galezowski, revealed the following alterations: 1st, the papilla is uniformly red over its whole extent, a phenomenon consecutive on capillary congestion; 2d, the *borders of the papilla* are effaced, blurred, on account of a diffuse *serous exudation* which extends along the vessels over the retina; 3d, the principal branch of the central artery, which is distended in the lower part of the retina, presents a fusiform dilatation, whilst near the papilla it seems to be in a state of spasmodic contraction. According to M. Galezowski: "There is reason to suppose that all these disorders are due to spasmodic contraction of the arteries in some places, and their dilatation in others. Hence the occurrence of papillary congestion in some parts, and of anæmia in others, a state of things resulting in peri-papillary serous infiltration." (B.) See also the case recorded by M. Bonnefoy, in 'Le Mouvement Médical,' 1873, p. 276. (Note to the First Edition.)

In all the patients affected by *hysterical amblyopia*, who were recently examined by M. Landolt at La Salpêtrière, the visual field for white and for colours was found to be concentrically diminished, even in cases where visual acuity and central perception of colours are normal in the eye of the non-anæsthetic side. All the functions of the retina of the eye, on the affected side, have proportionately decreased

[2] To the cases mentioned by M. Charcot, the following, noted in his wards, should be added, confirming as it does his teaching in every particular.

Berthe Chat—, aged 18 years and a half (July, 1873), was subject from childhood until her twelfth year, to epistaxis, always supervening in the *right nostril*; and from the age of twelve until she was fifteen, to cephalalgia, affecting her at monthly periods nearly. At fifteen, without any known cause, and irrespective of any appreciable hereditary influence, she had suddenly a convulsive seizure, with loss of consciousness. Rare during her sixteenth and seventeenth years, these attacks were multiplied in the course of her eighteenth year. Some of them, which belong to the category of simple hysteria, recur during every two or three months; others partaking of the nature of hystero-epilepsy appear every month, with tolerable regularity. The occurrence of the catamenia (in January, 1873) did not modify, in any perceptible manner, the frequency and character of the convulsions.

Does there exist any sign which would enable us to indicate, with certainty, the character of the case; to ascertain, for instance, whether the sclerosis has, or has not definitely taken up its abode in the lateral columns? I do not believe, gentlemen, that in the actual state of science, a single symptom can be mentioned which offers, in this respect, an absolute worth in prognosis.

Convulsive trepidation of the contractured members, whether purposely induced, or spontaneously supervening (*spinal epilepsy*), a certain degree of emaciation of the muscular masses, a slight diminution in the energy of electrical contractility, ought not, judging from my own observation, to make us altogether despair of seeing the contracture disappear, without leaving any trace behind. On the contrary, atrophy, limited to certain groups of muscles, especially if fibrillary contractions be added, similar to those we

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At the time of her admission to La Saltpêtrière (Sept. 1872), this young girl presented on her right side: 1st, complete hemianæsthesia; 2d, ovarian hyperæsthesia.

October 8.—After an attack, accompanied by delirium lasting for about twelve hours, contracture of the right lower extremity with talipes equino-varus supervened; the contracture is complicated by an almost constant tremulation (spinal epilepsy). From the 10th to the 25th of October, the situation is unchanged, in spite of the occurrence of a hystero-epileptic fit.

October 30.—Convulsive paroxysms, in which hysteria predominates. During the second paroxysm, the persons who held the patient lest she might hurt herself, felt the right leg, which till then had been in extension, become suddenly flexed upon the thigh, and when the patient came to her senses, the contracture had ceased. Chat— retained a certain degree of debility in the right inferior extremity, principally in the foot which was turned inwards.

November.—Berthe walks without limping: the right foot still turns inwards occasionally, and its point knocks, at times, against the left foot. Sometimes, also, the right leg is taken with a trembling which lasts five or six minutes, and which is followed by a sort of numbness that generally remains during the course of the day. "Then I can no longer feel my leg," says the patient.

1873.—The muscular debility has progressively diminished. To day (July 8) Chat— is as strong on one side of the body as on the other; the right hemianæsthesia and ovarian pain have not changed. This case is an additional proof that hysterical paralysis, with contracture, may suddenly disappear without the assistance of any intervention. (B.)

observe in progressive muscular atrophy, or a very marked decrease of faradic electricity, ought to make us suppose not only that the lateral columns are profoundly injured, but, also, that the anterior cornua of the gray substance have been invaded. I have not observed, up to the present, these latter symptoms except in cases of hysterical contracture of very old standing, and which left but little hope of ever again seeing the affected members resume their normal functions.

I will add, in conclusion, that the existence of a spinal organic lesion, of more or less gravity, will be placed almost beyond doubt if, under the influence of sleep induced by chloroform, rigidity of the members only gives way slowly, or even persists to any marked extent.

In my opinion, so long as these symptoms are not distinctly manifested, we should despair of nothing. It is besides important not to forget that *lateral sclerosis*, even when completely established, is far from being an incurable disorder, as I hope soon to prove to you.

In the case of the patients to whom I have called your attention, the contracture occupied either the whole of one member or of two members, or even more. But there are cases in which spasmodic rigidity remains limited to some portion of a member, as the foot for instance, when it produces a sort of *hysterical club-foot* (*talipedal distortions*, of M. Laycock). Quite recently, Dr. R. Boddaert communicated to the Medical Society of Ghent a most interesting case of this kind.[1] The contracture had occasioned the deformity, known as talipes varus. Similar cases have been collected and published by Dr. Little,[2] by C. Bell,[3] by Dr. F. C. Skey,[4] and by some other authors.

If it were not for certain reasons of propriety, I could, in my turn, gentlemen, relate in all its details the history of a case which resembles that published by M. Boddaert.

Let it suffice to inform you that a young girl, at present twenty-two years of age, very nervous, and belonging to a family in which nervous affections predominate, was, three years ago, suddenly seized with painful contracture of the muscles of the left leg; it could be assigned to no cause, and she had not previously shown any characteristic symptom of hysteria. This contracture, which made the foot assume the attitude of most marked talipes equino-varus, gave way to several remissions in the course of the first year, but during nearly two years it has remained stationary and seems permanent (June, 1870).

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[1] 'Annales de la Société de Médecine de Gand,' 1859, p. 93.

[2] A 'Treatise on the Nature and Treatment of Club Foot and Analogous Distortions,' London, 1839, Case 35.

[3] 'The Nervous System of the Human Body,' 3d Edition, 1836, Case 177.

[4] 'Hysteria, etc.: Six Lectures Delivered to the Students of St. Bartholomew's Hospital,' 1866, 3d Edition, London, 1870, p. 102.

Several of the muscles of the leg have become greatly atrophied; they likewise present very marked fibrillary contractions, and respond but feebly to electrical excitation. Hence, I believe that there is little chance of seeing the contracture become resolved, more especially as it shows but very imperfect amendment during sleep, induced by chloroform. I will also point out a most interesting peculiarity, from a clinical point of view: this young girl has experienced hysterical seizures in the course of the last few months only.

LECTURE XIII.

HYSTERO-EPILEPSY.

SUMMARY.—Hystero-epilepsy. Meaning of this term. Opinions of authors. Epileptiform hysteria; hysteria with mixed crises. Varieties of hystero-epilepsy; hystero-epilepsy with distinct crises; hystero-epilepsy with combined crises, or attaques-accés (seizure-fits). Differences and analogies between epilepsy and hystero-epilepsy. Diagnostic signs supplied by examination of central temperature in hystero-epileptic acme, and in epileptic acme. Epileptic acme; its phases. Clinical characters of hystero-epileptic acme. Gravity of certain exceptional cases of hystero-epilepsy. Case recorded by Wunderlich.

GENTLEMEN,—In the brief clinical description which I gave you, in reference to each of the patients who had passed under your observation at our recent conferences, I studied to bring out the principal characters presented by the convulsive seizures to which they are subject.

You have been able to recognize, with ease, that we have not here to deal with common attacks, which can be assigned at once and without discussion to the classic type. Nor is it merely by their great intensity that these convulsive phenomena are distinguished, but also by the form they assume; and what most strikes the observant witness is to find amongst the clonic convulsions of hysteria, certain more or less marked features which recall the phenomena of *epilepsy*.

In point of fact, the convulsive form of disease which is found in all these cases, is that which has been designated, in these latter times, by the name of *hystero-epilepsy;* and, remember, it is the only form met with in these patients. These women would not, therefore, be simply hysterical patients, they are all *hystero-epileptical*. In what respect do they differ from ordinary hysterical patients? This is a question concerning which it is important to have a clear understanding, and in order to secure that object, I request your permission to treat the matter at some length.

I.

If we keep to the terms of the denomination generally employed —*hystero epilepsy*—it would appear as if no misunderstanding could arise. It signifies that in patients, so affected, hysteria is present in combination with epilepsy, so as to constitute a mixed form, a kind of hybrid composed half of hysteria and half of epilepsy. But does this appellation, in reality, accurately interpret the phenomena? Superficially looked at, it would seem to do so, since we have recognized in the seizures some of the features of epilepsy. This, in fact, is the manner in which most modern authors appear to understand the term. According to their view, hystero-epilepsy would be a mixture, a combination of the two neuroses, varying in proportions in different cases; it is not epilepsy alone, nor hysteria alone, but both together.

Such, I repeat, is the most popular doctrine. However, it is far from being universally accepted, and the camp of its adversaries still reckons many adherents. These refuse to admit the legitimacy of this hybrid, half-epilepsy, half-hysteria. They do not, indeed, deny that epilepsy and hysteria may coexist in the same individual. The most superficial observation would protest against any such assertion. There is nothing to authorize the belief that these diseases are antagonistic, and it might even be possible, though it has not been proved, that patients affected by one of them, might by that very fact be predisposed to contract the other. But, under such circumstances, it is added, the convulsive accidents remain distinct and separate, without exercising reciprocal influence over each other, in any marked manner, and, above all, without mingling confusedly so far as to justify the creation of a mixed intermediate species, in one word, of a *hybrid.*

What, then, according to this view, is the signification of those attacks, the existence of which is so clearly established by the very cases that form the foundation of our study, and in which epilepsy seems mixed up with the ordinary symptoms of convulsive hysteria?

Epilepsy would, in their opinion, be present here only in the external manifestation; it would not be substantially existent. In other words, we would have, in these cases, hysteria solely and always present, taking on it the semblance of epilepsy. The term *epileptiform hysteria*, which, if I err not, Louyer Villermay was one of the first to employ, would serve to designate these mixed attacks. The convulsion, epileptic in form, would here appear, as it appears in so many other affections of the nervous system, as an accessory element, without altering in anything the nature of the original disease.

II.

That, gentlemen, is the thesis to which I give my entire adhesion. It has already been maintained by some most competent authorities. Of them, I may cite Tissot, Dubois (of Amiens), Sandras, and M. Briquet, who are very explicit on this question. "Hysterical seizures," says M Tissot, "sometimes closely resemble epilepsy. Hence, they have been classed as a particular form of hysteria, under the name of *epileptiform hysteria.* But, nevertheless, these seizures have not the true characters of epilepsy."[1]

M. Dubois (of Amiens), considers epileptiform hysteria, as hysteria with an extra degree of intensity superadded to its symptoms,[2] Sandras expresses a similar opinion.[3]

M. Briquet, whose article on this subject bears the mint-mark of the soundest observation, says that this species of *hysteria, with mixed attacks*, is only a particular form of hysteria—is simply very intense hysteria,—the prognosis is not essentially modified: the nature of the cause which occasioned the hysteria and certain conditions special to the affected individual, account for the modifications observed in the form of seizure. The nature of the hysteria is not, itself, radically altered.

Be good enough to remark, gentlemen, that this is something more than a mere question of words; it is a question also of nosology, and consequently, a question of diagnosis and of prognosis. These circumstances will, I trust, suffice to justify in your eyes the details on which I am obliged to enter, in order that the conviction which I entertain may take its place in your minds.

III.

Let us, therefore, inquire upon what basis the prevailing doctrine reposes. Hysteria and epilepsy, it is alleged, may be combined in different ways in the same patient. M. Beau, who studied in this hospital, states that he found this combination in 32, out of 276 patients. It takes place in different modes, and the following categories may be legitimately established.

A. In the first group, the hysterical seizures and the epileptic fits remain distinct; this is what M. Landouzy proposes to call *hystero-epilepsy with distinct crisis.* Well, gentlemen, that would be the most frequent form, seeing that 20 out of the 32 cases reported by M. Beau belong to it. Two subdivisions, however, should be established in this species:—

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[1] Tissot. 'Maladies des Nerfs.' t. iv, p. 75.

[2] Dunont, 'De l'Hystéro-Epilepsie.' p 11.

[3] Sandras, 'Maladies Nerveuses,' t. i, p. 205.

1st. Epilepsy is the primary disease: upon this stock hysteria becomes grafted in due time, that is to say, most frequently at the period of puberty, under the influence of certain causes, and of moral emotions in particular.

A case which M. Briquet quotes from Landouzy deserves to be summarized for your instruction as bearing upon this point. A young woman, who had been affected with epilepsy from her childhood, got married at the age of eighteen. The disease, which she had concealed, soon showed itself. Hence arose vexatious disputes which engendered hysteria. The attacks, proper to the two neuroses, were separate and preserved their specific characters, without either being influenced by the other. A reconciliation having taken place on account of her pregnancy, between the patient and her husband, domestic peace was re-established which caused the hysteria to cease, but the epilepsy persisted.

2d. At other times, epilepsy is superadded to hysteria. This condition appears to be much rarer than the preceding. M. Briquet, however, reports a case which came under his own observation in which the attacks were distinctly separate. The mind becomes obscured, in the long run, in patients belonging to this class, owing undoubtedly to the influence of the epilepsy.

3d. Some other combinations, of a secondary order, have been mentioned. Thus:—

a. Convulsive hysteria coexists with minor epilepsy [1] (Beau, Dunant).

b. Convulsive epilepsy is superadded to some of the phenomena of non-convulsive hysteria, *e. g.*, contracture, anæsthesia, etc. We have a case of this kind among our patients.

But these different combinations alter nothing in the essence of things. Most frequently the two diseases, in hystero-epilepsy, exist simultaneously and proceed their several ways, without reacting on each other in any serious manner, each of them preserving its own characteristics and proper prognosis. With respect to this first form of hystero-epilepsy all authors are agreed. The second form only is concerned in the debate.

B. In this form, the *hysteria and the epilepsy are coeval;* they both develop at the same time. Here the crises do not remain distinct; they have never been so. From the outset, the intermingling had been effected, and, in subsequent attacks, the two convulsive forms will always show themselves combined, though in varying proportions, without being ever, at any moment, completely dissevered.

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[1] The *petit mal* of French authors. This form of the disease, so distinct from the common form, to which the name epilepsy is popularly applied, and yet so important in itself, especially when questions of hereditary predispositions arise, seems to deserve a distinct designation. (S.)

To this condition the name of *hystero-epilepsy with combined crises* has been given. In the technical jargon long employed in the special wards of La Salpêtrière, these crises, in such cases, are called "*attaques-accès*" (which we may translate "seizure-fits").

IV.

Is there really any *epilepsy* in these mixed crises? Such is the question which we have now to discuss. With this view, it is right that we should take the description of hystero-epilepsy with mixed crises, as agreed upon by authors, and examine it under all its aspects. From M. Briquet, in especial, I borrow the description of the seizure-fit. It seems to me to be in complete concordance with the results of my own observation.

a. From the outset, the mixed attack assumes its proper character; from that moment, it is epileptiform hysteria. I would recall to your memory the patient Etch—, who, in her first attack, fell into the fire, and injured her face.[1]

b. The hysterical aura, such as we have described it, always constitutes a premonitory symptom. This aura, generally of long duration, occupies the abdomen, the epigastrium,—at all events, it does not affect the head alone from the very first, nor one of the extremities, as takes place in epilepsy with aura. Hence it is perfectly exact to say that patients suffering from hystero-epilepsy with mixed crises are nearly always forewarned in sufficient time to enable them to take precautions or to seek a place of refuge, when the fit is coming on.

c. In the convulsive attack, the *so-called epileptic phase* generally presents itself first, to open the scene. The drama begins—a sudden shriek, extreme pallor, loss of consciousness, a fall, distortion of the features—then tonic rigidity seizes on all the members. This rigidity, remark it well, is rarely followed by the clonic convulsions, brief in duration, limited in oscillation, predominating on one side of the body, such as we see them in true epilepsy. Nevertheless, the face may become greatly tumefied and violet-colored. There is foaming at the mouth, and the foam is sometimes bloody on account of the tongue or lips having been bitten. Finally general relaxation of the muscles may follow, with coma, and stertorous respiration during a less or greater length of time.

d. To this first phase, which I repeat is the one chiefly concerned in the dispute, the *clonic phase* succeeds. Then all is hysteria: great gesticulations, having a purposive character, supervene, and sometimes violent contortions are made, characteristic of the most various passions, such as terror, hatred, etc.[2] At the same time *paroxysmal delirium* breaks out.

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[1] Lecture XI. This patient is also mentioned in Lecture IX.

[2] See *ante*, figures 19, 20, and 21.

e. The termination of the attack is marked by sobs, tears, laughter, etc.

These different phases do not always succeed each other in so regular a manner; they get entangled occasionally, and now one, now the other predominates. In the patient C—, for instance, the tonic phase prevails to a great extent over the other, and sometimes it is almost exclusively manifested.

V.

We have arrived, gentlemen, at the critical point. In what does this hysteria with complex crises differ from ordinary hysteria, if it be really separate? In what does it resemble true epilepsy, if there be reason for such an approximation?

Is the appearance of tonic convulsions a novel and unwonted fact, in the classical description of the common hysterical attack? Certainly not. It is not really exceptional, in common hysterical attacks (when no one thinks at all of interpolating the epileptic element), to see the supervention of tonic convulsions occur, having an epileptiform character, especially at the beginning of the seizure. All authors are agreed upon this point. These convulsions are occasionally so marked that M. Briquet has been induced to establish, side by side with the clonic or classic hysterical seizure, a species of seizure in which *semi-tetanic stiffness* predominates in the body and members. Does it not, therefore, seem already probable that the so-called epileptic form is, properly speaking, only the exaggeration, the highest degree of development of this *variety* of common hysteria?

VI.

If, on the other hand, we turn our gaze upon true epilepsy, we shall meet with a certain number of characteristic peculiarities of which we can easily make profitable use.

We should point out, in the first place, that, according to the description already given, the epileptic type is never represented in the seizure fits, save in an imperfect manner, in rough outline as it were; but, indeed, that alone would not be a decisive argument. Here is a more significant character.

Never, in descriptions of hystero-epilepsy with mixed attacks, do you find mention made either of the *petit mal*, or of the *epileptic vertigo.* We might also add, as supplying material for an important distinction, that, in this form of hystero-epilepsy, even the most intense epileptiform attack is judging from our own observation modified and sometimes even arrested in its development by *compression of the ovary.* This never happens in true epilepsy, as we have over and over again assured ourselves by experiment.[1]

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[1] V. *ante*, Lecture XI.

In cases of mixed attacks, even when frequently repeated, it is acknowledged by authors, that obnubilation of the intellect and dementia are never the consequences of these seizures. This is contrary to what would almost necessarily follow, if epilepsy were really in question. I cannot do better, in connection with this, than recall to your mind the case of the patient Ler—, who, for nearly forty years, has been subject to the most violent epileptiform hysteria. This woman is, no doubt, odd, and whimsical in her ways, but her intellect remains what it was at the outset. The information we have received, on inquiries made, do not permit the survival of any doubt as regards this fact. In short, in cases of this kind, the prognosis is nothing different from that of intense hysteria. Such is likewise the opinion of M. Briquet.

From this consideration a practical conclusion is deducible, well calculated to command your attention.

There is, lastly, another characteristic on which I beg leave to dwell at some length, because it has not hitherto been noted, so far as I am aware, and because, in my judgment, it is decisive. This characteristic is yielded by thermometrical exploration; and I hasten to seize the opportunity which presents itself now of showing you, by a new example, the advantage which may be derived from this mode of investigation in the clinical treatment of diseases of the nervous system.

It is not, gentlemen, that the tonic epileptiform convulsions of hysterical patients differ, in any respect, from the convulsions of the epileptic attack, so far as changes of central temperature are concerned. The tonic hysterical seizure, if it have but a certain intensity, raises the temperature by 1° C. (=1 8° F.), nay, by a degree and some tenths (38°, 38.5° C.=100.4° F., 101.3° F.), exactly as we find to result from an attack of true epilepsy. This is a fact the accuracy of which we have had many opportunities of testing in these wards.[1]

But if, as regards thermic elevation, the attack of epileptiform hysteria and the attack of true epilepsy be identical, it is quite otherwise when we have to deal with those fast following fits that constitute what, as regards epilepsy, have been called in France *les series* or *etat de mal* (= *status epilepticus*,—which we may translate by the term *epileptic acme*).

Of this *epileptic acme* we can distinguish two kinds: the *minor acme*, (*les petites series*), constituted by from 2 to 6 fits, and the *major acme* (*les grandes series*), in which from 20 to 30, or even more fits, have been reckoned in the twenty-four hours. I address myself exclusively to the latter, because the phenomenon, on which I wish to lay stress, then manifests itself in its typical state of full development. In such cases, gentlemen, that is to say, when a great number of true epileptic fits succeed each other, within a brief space the central temperature becomes remarkably augmented; and, most assuredly, this thermic increase cannot be attributed exclusively to the repetition, any more than to the intensity, of tonic muscular contractions, for the convulsions may completely cease for several days, whilst the temperature nevertheless persists, during this time, at a very high elevation.

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[1] Bourneville, 'Études Cliniques et Thermomètriques sur les Maladies du Système Nerveux.'

We can observe and follow these peculiarities on the diagram which I place before you, and which represents the changes of central temperature in the patient Chevall—, during the course of the *epileptic acme* which she has recently experienced. (Fig. 25.)

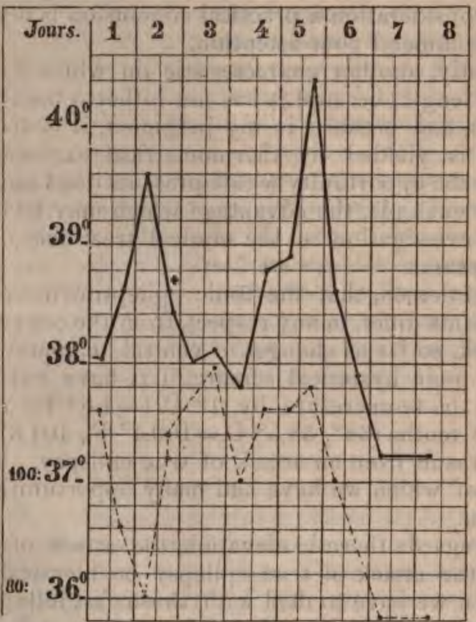

FIG. 25.—Temperature (C.) taken shortly after the eleventh fit. From the evening of the first day until the morning of the second, thirty-one fits occurred. + Temperature after a remission of four hours. After this the fits take place at greater intervals, and cease on the third day. The dotted line represents the state of the pulse.

It must be borne in mind that this elevation of temperature is, in the great majority of cases, even after complete cessation of the convulsions, an omen of the darkest significance. It is, besides, most frequently accompanied by a general state of the constitution which, of itself, gives much cause for apprehension. Thus, sometimes, a more or less marked delirium exists, which M. Delasiauve attributes to *meningitic congestion;* sometimes, on the contrary, a more or less profound coma—the *apoplectiform congestion* of authors —is found. In both cases we observe great prostration, dryness of the tongue, tendency to rapid sloughing over the sacrum; lastly, occasional production of transient hemiplegia, the cause of which has not, as yet, been revealed by any post-mortem examination.

However, and this is a most important datum to note, this elevation of temperature, even when it exceeds 41° C. (=105.8° F.), and is accompanied by the grave symptoms just enumerated, is not to be regarded as a sign *necessarily* heralding a fatal termination. You perceive by the record of Chevall—, that a patient may still recover, even from the midst of all these grievous circumstances. Augmentation of temperature above 41° C. (=105.8° F.) is not, therefore, necessarily *terminal* in such cases; consequently, the assertions published by Herr Wunderlich, and after him by Herr Erb, in relation to this point, must be subjected to abatement.[1]

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[1] The case of the patient Chevall— is related at full length, up to 26th March, 1872, in our 'Études Cliniques et Thermométriques sur les Maladies du Système Nerveux' (Obs. xxxiii, p. 285). Since that period, Chevall— Edmée has been seized with new accidents issuing in a fatal termination. We think it all the more useful to relate them here because, besides completing the former record, they supply additional proof in corroboration of the opinions stated by M. Charcot in the present lecture.

1873, *February* 9.—For about a week Chevall— has been tetchy and irritable; sometimes she has been so violent that constraint was necessary (maniacal excitement).

February 10.—Last night the agitation augmented; Chevall— prevented the other patients from sleeping, by her cries. She, however, became calm after three o'clock A. M. Three fits were noted during the night. From one o'clock P. M. till three o'clock P. M. the fits multiplied. At three o'clock: pulse 100; rectal temperature, 38.6° C. (=101.48° F.).

February 11.—Yesterday, from one o'clock till nine o'clock P. M., forty-three fits were counted; and from that until seven o'clock A. M., seventy fits; from seven o'clock till eleven o'clock A. M., when this note was taken, there occurred thirty-five fits. The following is a description of the fits:

Five or ten seconds before their occurrence, the pupils (especially the left) became widely dilated. Sometimes, in addition, we have little complainings, grinding of the teeth, and, exceptionally, a slight cry. The fit begins: the eyeballs are subjected to very marked convulsive movements (nystagmus); the face grows pallid, and is deviated to the left; the gaze, at first fixed and direct, is averted to the left. The left arm rises, and then stiffens, simultaneously with the right, which, however, rests upon the bed. The tetanic stiffness next invades the lower extremities. At the end of a few seconds, we notice semi-occlusion of the left eyelids, which are agitated, like the muscles on the same side of the face, by rapid convulsive movements.

Ten to fifteen seconds after, the face and eyes turn to the right; the body inclines to the right; the left eyelids open, and remain nearly motionless; but, to make up for this, the convulsions seize upon the right eyelids and the muscles of the right side of the face. The mouth, at first drawn to the left, is now drawn to the right. The clonic convulsions manifested during this phase, which had at first invaded the members of the left side, now predominate on the right.

Finally, the fit concludes by snoring, extreme lividness of the face, and foaming at the mouth. At the close of the fit, the pupils resume their normal dimensions.

During the remissions the patient is in complete resolution. When raised and let go the limbs fall inert. Energetic pinching provokes a slight raising of the left

I should remind you, in passing, that this rapid increase of temperature is far from being the exclusive appanage of the epileptic acme; it is likewise observed in the so-called congestive, apoplectiform, or epileptiform attacks of progressive general paralysis, as was first pointed out by Herr Westphal, who has, however, offered an interpretation of the fact which is little in conformity with the reality.[1] It is also observed in the very similar attacks which may

arm, but nothing in the right. When the soles of the feet are tickled, reflex movements are set up, which are more intense on the left than on the right. The right eye is not injected, whilst considerable hyperæmia of the lower half of the left eyeball and a lesser vascularization of the lower lid exist. The nostrils are pulverulent. The digestive tube presents no particular symptoms; there was a stool after enema yesterday; Ch— micturates in bed. Erythematous patch on the left gluteal region, profuse perspiration, augmented at intervals. At eleven o'clock: pulse 120; respiration 49, noisy; rectal temperature, 40.8° (=105.44° F.). At noon pulse 130; respiration 60

Six o'clock P. M.—Seventy-six fits have been noted since eleven o'clock A. M. of which thirteen occurred after half-past four o'clock P. M. Respiration 60; rectal temperature, 41.3° C. (=106.34° F.). Copious perspiration over the whole body, on both sides indifferently. The entire left side of the body (face, trunk, &c.) is plainly warmer than the right.

The eyelids are half open; the eyes turned up; the pupils are moderately dilated (the right still the more enlarged). Before every fit *dilatation of the pupils* augments in a remarkable manner. The *nystagmus* seems to appear almost simultaneously. Neither vomiting, nor stools, nor micturition Same condition of right gluteal region. Coma. Stertorous breathing.

Eight o'clock P.M.—Pulse—; respiration 70; rectal temperature 41.2° C. Fourteen fits. From this time forth the patient had no more fits. She died at three o'clock in the morning. Vaginal temperature (taken by another) was 41.2° (=101.10° F.). At eleven o'clock in the morning on the 11th February, *i. e.*, eight hours after death, rectal temperature 40° (=104° F.) (the corpse remaining in the bed). The pupils are moderately dilated, and both equally. Numerous livid stripes or wheals on the belly, back, buttocks, and thighs.

Post-mortem, Feb. 18.—The bones, dura mater, and sinuses present nothing abnormal. The quantity of cephalo-rachidian liquid is not augmented. Sanguineous suffusion on the convex surface of the cerebral hemispheres, especially on the right. Arteries, at the base of brain, healthy. Weight of encephalon, 1360 grammes. The pia mater is very slightly injected at base of brain; most marked at the sphenoidal lobe. The pia mater is easily detached on both sides, and the brain is equally moist.

Right hemisphere.—It weighs 5 grammes (=77.17 grs.) more than left. On certain convolutions, chiefly those lying adjacent to the Sylvian fissure, we note a hortensia coloration, some little abrasions, and, on a few, a very fine red punctuation. The convolution of the cornu Ammonis presents a very evident induration. This induration, which ascends interiorly along the said convolution, predominates at its extremity.

Left hemisphere —The cornu Ammonis presents an induration much less marked and circumscribed at its extremity.

Nothing notable in the cerebellum and isthmus.

Spinal cord.—The gray substance, viewed with the naked eye, seems a little deformed.

Thorax.—Considerable congestion of the lower half of the lungs. There is, moreover, a focus of red, recent hepatization in the lower lobe.

Heart, stomach, spleen.—Healthy; no ecchymoses. *Liver*, not congested. *Kidneys*, anæmia of cortical substance; pyramids distinct. *Bladder*, nothing. *Uterus*, fair size; recent corpus luteum on one of the *ovaries*: small cysts on the other (B.).

[1] Westphal, *loc. cit.*

supervene in the course of disseminated sclerosis,[1] and, lastly, in the attacks accompanied or not by convulsions which take place in cases of old cerebral foci (hemorrhage or ramollissement) or of cerebral tumors, whatever their origin. This thermic increase contrasts, in a remarkable manner, with the initial decrease which almost always exists at the moment of the formation of a cerebral hemorrhagic focus,—and that, as I have demonstrated, is a characteristic which may be profitably used in making a diagnosis.

But it is time to return to epileptiform hysteria, from which this digression has somewhat separated us. Complex fits are observed in hystero-epilepsy precisely as in true epilepsy. Landouzy speaks of one hysterical patient who had up to 100 fits a day. The *hystero-epileptical acme* may, besides, be prolonged over a considerable space of time. Georget quotes the case of a woman who suffered from an almost continuous succession of fits extending over forty-five days.

In the case of our patient Co—, whose seizures bear such a predominant and strongly-marked epileptiform character, the *paroxysmal acme persisted over two months*, and, at times, the accidents attained the highest degree of intensity. Thus, to mention one instance, on the 22d of January, the epileptiform convulsions followed each other, without interruption, from nine o'clock in the morning until eight in the evening; from eight until nine o'clock there was a resting space, after which the atacks came on again, as though with renewed vigour, and persisted, without the least lucid interval, for about the same length of time. We may without any exaggeration calculate that in round numbers she experienced from 150 to 200 epileptiform fits in the space of a day at that period.

Does not the persistence of such a state, without a fatal termination, already indicate by what an abyss true epilepsy is separated from hystero epilepsy? "If that were not hysteria," said the experienced head nurses of these wards, speaking of Co—, whose seizures they watched, "if it were really epilepsy, the woman would have succumbed long ago." This remark is thoroughly well-founded and perfectly correct.

Well, gentlemen, here is the point upon which, especially, I wish to lay stress,—never, during that long convulsive period, was the *rectal temperature* modified in a perceptible manner, in the case of Co—; on an average it stood at 37.8° C. (= 100.4°F.), it only rose to 38.5° C. (= 101.3° F.), in a quite exceptional and transient manner. (Fig. 26.)

I should add that, during all this time, the general condition of the patient never gave us the least uneasiness, in spite of the insufficiency of her alimentation and the enormous amount of muscular force she must have expended. Again her mental condition was, by no means, so deeply modified as it would necessarily have been if the disease had been true epilepsy. At no period was there involuntary evacuation of urine or feces. In the brief respites allowed her by the fits, the patient rose to satisfy the requirements of nature. In these intervals, also, which were in truth very brief, the hysterical character of the disease showed itself in all its fulness, especially during the first weeks. A flower twined in her hair, fantastic curls, an old bit of mirror attached to the bed-post, —these things sufficiently attested the favourite occupations of this woman during her intervals of ease.

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[1] V. *antè*, Lecture VIII.

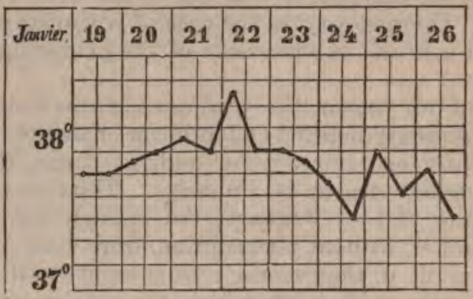

FIG. 26.

But I desire, above all, to call your attention to the thermal state which careful investigation enables us to discover. It would, in short, follow from what precedes that, whilst, in the *major acme of epilepsy*, the temperature rises very rapidly to a high degree, and the position of affairs becomes extremely critical,—in the *major acme of hystero-epilepsy* on the contrary, the temperature rarely exceeds the normal standard, whilst the concomitant general state of the patient is not of a kind to inspire uneasiness. It is not necessary, I think, to dwell at any length upon this topic in order to emphasize so striking a contrast.

I do not, however, gentlemen, want you to take in its strictly literal meaning the last term of the proposition which I have just enunciated; it undoubtedly applies accurately to the great majority of cases, but the chapter of exceptions exists. It would not, in fact, be unexampled to find hysteria terminating in death during the convulsive phase. It is true that this sad result is nearly always owing to a peculiar kind of attack, the *dyspnœic seizure.*[1] But I repeat, the convulsive fits may themselves be the cause. As an example, I may remind you of a case of this character published by Herr Wunderlich.[1] It relates to a case of hystero-epilepsy, comparable in many respects to that which I have just described.

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[1] Briquet, *loc. cit.*, pp. 383 et 538

[1] The following is a translation (after M. Teinturier) of Wunderlich's case to which M. Charcot alludes:

NOTE.—*Eight weeks of apyretic hysteriform convulsions, without apparent danger. Sudden and deplorable change, without augmentation of paroxysmal intensity. Death in the course of a few hours, with a temperature of* 43° C. (=109·4°F.)

Anna Vogel, æt. 19, servant, had twice menstruated in the fortnight before falling ill, but otherwise in good health, was taken with convulsions, for the first time, on the 13th August, 1855, after a severe scolding, according to her own account. The convulsions came on again, in the evening of the 17th, and in the morning of the 18th, and continued almost uninterruptedly throughout the night, from the 18th to the 19th. Admitted at noon on the 19th. She presented at midnight slight subsultus in the left arm, in which the presence of paralysis without insensibility had been noted. Then she experienced a feeling of anguish (constriction in the epigastrium), gave a slight cry, and had convulsions, first in the left half of the face, and then in the right also; the mouth was open, the eyelids were alternately opened and closed, the eyeballs turned greatly upwards. Then supervened violent and rapid clonic convulsions in the lower extremities and pelvis, by which these parts were projected forward, backward, and sideways. The face was cyanosed, and foam flowed from the mouth. At the end of a minute, deep and superior breathing; relaxation of the limbs and face. Afterwards sleep, apparently peaceful; lastly, yawning, opening of the eyes, and return to consciousness after six minutes.

The patient is in good condition, her tongue is little loaded; the temperature is 38.12°C.(=100.6° F.), the pulse 140 (after the fit); nothing abnormal. She says, however, that she cannot move her left arm, and requests that it shall not be touched, because otherwise she will get convulsions. Nevertheless, she can grasp strongly with the left hand.

In the night of the 19-20th six seizures; and in the day following, seven seizures. No albumen in the urine; considerable uric sediment. Tongue loaded. Temperature, morning and evening, 38.12° C. (=100.6° F.); pulse 132; respiration 24—32. In the night of the 20-21st, seven seizures; thirteen seizures till the morning of the 22d. Temperature, 37.76° (=99.96°F.); stools normal; urine slightly turbid from the presence of albumen.

From eight to sixteen fits a day, in the following days. Condition otherwise tolerable; no marked elevation of temperature, which is generally normal, never above 38.12° C. (=100.6° F.), except one evening, when it reached 38.75° (=101.75° F.); pulse usually above 112; tongue loaded. On the 16th, confluent vesicular miliary eruption on the finger tips. Urine charged with phosphates, without albumen. During the seizures she sometimes loses consciousness, sometimes not; occasionally shrieks greatly. Sensibility persists in the left arm and leg.

7th September.—The fits become more frequent, last several days without interruption; during the seizures she often talks and shrieks. Frequent evacuations of urine and feces in bed. Improvement, then stationary condition until the evening of the 2d October, when the patient presents a marked attack of collapse. In the night of the 3d no particular seizures. In the morning agitation of the arms and divergent strabismus. The head inclined forward and to the left; consciousness preserved; slight cyanosis. From ten o'clock forth, deglutition impossible; at noon, trismus; at a quarter to two o'clock, strong convulsions, not affecting the head; pulse extremely frequent; temperature, 41.87°; intense cyanosis, foaming at the mouth; tracheal râle. Died at a quarter past two o'clock; temperature, 43.1° C. (=109.58° F.). A quarter of an hour afterwards, temperature 42.75° C. (=108 95° F.).

Post-mortem.—Body in good condition; large cadaveric spots in the lower part; no muscular rigidity. The cranium and its contents gorged with blood; posterior convolutions slightly flattened; cerebral substance rather hard. Slight turbid

The patient in question experienced epileptiform attacks for more than eight weeks, though in number they were somewhat limited, and not accompanied by any marked augmentation of temperature when suddenly—without known cause, without the intervention of new accidents—the scene changed two days before death; the patient fell into collapse, and in a short space of time the temperature rose to 43° (= 109·4° F.).

This example, gentlemen, will suffice to show you that, in presence of a case of hystero-epileptic acme, whatever be its intensity, or however great the chances of a favorable issue, it would be imprudent to abandon ourselves to a feeling of complete and absolute security.

thickening of the *pia mater* at the base of brain. Capacity of cerebral cavities nearly normal, parietes usual consistence. *Pons* and *medulla* injected with blood, dirty grayish red. *Lungs* congested and œdematous. *Heart* normal. *Liver* fatty here and there, exsanguine; bile, clear and dark brown. *Spleen*, small, soft, pale brown, exsanguine. *Stomach* dilated, otherwise normal, as were the intestines. *Kidneys* greatly gorged with blood: concretion size of half a pea in calyx of left kidney. *Uterus* normal. *Ovaries* containing numerous cysts, as large as peas (Wunderlich, 'Archiv der Heilkunde,' t. v, p. 210).

APPENDIX.

CASE OF PARALYSIS AGITANS.

Antecedents.—Probable cause of disease. Commencement; the members successively invaded by debility. Tremor of the head, then of the limbs.

State of the patient in 1874; *general attitude. Tremor. Gait: propulsion and retropulsion. Temperature, pulse, etc.*

Modifications supervening in the disease, from July, 1874, *until July,* 1875.

Gav— Annie Marie, æt. 62, was admitted to La Salpêtrié, December 31st, 1872; came under M. Charcot's charge (Salle St. Alexandre, No. 3), on the 12th November, 1873.

Antecedents.—Her father, a carpenter, died of an accident when she was only twelve years of age. Her mother, who succumbed at the age of seventy-four, was of a nervous disposition, easily moved to passion, but had been affected neither by tremor nor by paralysis. Her only sister died of pleurisy at forty. None of her relations, so far as she was aware, suffered from nervous affections nor, particularly, from tremor.

Gav— arrived in Paris at the age of four. Her childhood and youth passed without the occurrence of any incident worth noticing. From the age of fourteen she menstruated regularly. Having married at the age of twenty-eight, she has had five children. Pregnancy and confinement generally favorable. Of the five children, the eldest (a boy), died during the Commune, aged 35; the second and third (boys also) enjoy good health; the fourth, a female, aged 28, is subject to nervous attacks at long intervals; the fifth died at birth.

The patient assures us that she has never had any serious illness; never, for instance, was affected by rheumatism or chorea. Although she had been a costermonger for thirteen years, she never gave way to excess in drink. She has always lived in healthy lodgings, well exposed to the sunshine; she was happy in her home, and never suffered from any privations.

Invasion of the disease.—Her affection first showed itself in 1868, under the following circumstances. Her third son, of whom she was particularly fond, unexpectedly told her one day that he had enlisted as a soldier. This news greatly afflicted her; she wept much over it, and from the following day was aware of a weakness in her right arm. Soon after, the left arm was taken in the same way, then the right lower extremity was invaded, and (simultaneously) the left. During the night-time she had cramps in her legs which made her cry out. Next she experienced weakness in the loins. At the time of her entrance into hospital (December, 1872) she was not so weak as at present (July 8th, 1874). The trembling began to invade the members, affecting first the right upper extremity, in the early months of 1873. Lastly, she noticed about the same time that she was affected by retropulsion; one day, having missed her step, she felt herself compelled to walk backwards in spite of herself.

Present state (July 8th, 1874).—The general attitude of the patient, when standing, is that described by M. Charcot in Lecture V. The body and head are bent forward; the neck is stretched and the head seems as though fixed on a rigid stem. The features are quite immobile; the brow is but faintly marked with wrinkles; the eyelids are moderately open; the patient can, however, raise or lower them with ease. The eyes, but slightly expressive, are directed forward; the patient must turn her whole body in order to look aside. Sometimes the lips adhere, but generally the mouth is half open, the lower lip, relaxed and hanging, allows the dental arch to be seen, the saliva flows involuntarily. The lips and tongue do not tremble. Deglutition, it appears, is almost always laborious.

The arms are held slightly apart from the chest; the fore-arms, half flexed, are so placed that the hands rest on the umbilical region, whilst the elbows are somewhat apart from the body. The thumb, slightly inflexed, rests usually on the forefinger; the other fingers are slightly bent and gathered together. Both hands are disposed in the same manner.

The legs approximate, though the knees do not touch. If the legs are separated, the equilibrium becomes uncertain. Whether the eyes are open or closed the attitude of the patient is the same.

She sits down heavily, and all of a sudden. She cannot rise until she get assistance, and even then the attendants have to use strength to raise her. After hesitating, she begins to walk, advancing first with short steps, afterwards hurriedly,—there is *propulsion*. "Sometimes," says Gav—, "I am driven very far, until I meet a wall,—and if I don't, I fall." The existence of *retropulsion* is also marked: to observe it, it is only necessary to pull the patient gently by the skirt,—the method adopted by M. Charcot. Immediately she begins to walk backwards, and with such rapidity that she would soon fall if not watched. The hesitation of the patient, before returning, is greater than before beginning to walk.

The trembling is scarcely noticeable, especially when she is at rest. The head trembles, at times, a little more than the hands. When these are hanging by the side, they generally remain motionless. The patient can incline her head more than it is usually inclined, but it is impossible for her to raise it in extension completely, because "the vertebral column is stiff."

Neither cephalalgia, nor vertigo, nor giddiness. The intellect is preserved and the memory good. Her *sleep* is less abridged than we find to be the case with most patients suffering from the same complaint. She would even sleep well if she were not frequently waked up by pains in the heel "that prick me, and you 'd think 'twas water running inside the heel." She complains of a constant feeling of heat, and only keeps a sheet over her even in winter time.

We have mentioned the state of her strength, measured by the dynamometer (Note to Lecture V) and that of her temperature, so we shall not revert to these points.

July, 1875.—The *weakness* has gone on increasing. The general *attitude* remains the same; however, the head and trunk are more and more inclined forward; besides, a sort of *lateral inclination* has occurred, in consequence of which the right half of the body precedes the left half in walking.

At present, the *lips* are almost always adherent each to the other; the upper lip is puckered; sometimes, according to the patient, both of them are stiff. The dental arches are not pressed against each other. It would seem as if the patient drew her lips together to diminish the trembling of the chin; in spite of this precaution, her legs are stirred by little twitching movements, which, to use her own words, remind one of the motion of rabbits' lips. The *tongue* trembles even within the mouth; when protruded, the trembling is augmented.

The *trembling of the head* is composed of antero-lateral, and sometimes lateral, shakes, of very limited extent. These oscillations are transmitted to the head by the body, as was mentioned in the course of the lecture. When the patient is sitting, her *legs* tremble, and her feet beat rapidly on the floor, striking little blows. To sum up:—trembling has progressed, so far as the head and lower extremities are concerned; but in the upper extremities there is little alteration. Let us also notice the necessity of changing position (fidgetiness), which was little marked in 1874, and was only experienced during the day, is much more manifest at present, and torments the patient not only during the daytime, but whilst reposing in bed (at night). Her sleep is less than it was formerly. The patient still takes exercise in the ward and in the courtyard of the infirmary (B).

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